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PHYSIOLOGICAL MECHANISMS IN HEART FAILURE.[1]

By B. C. SINCLAIR-SMITH,
Sydney.

IT may be said that a heart fails when it ceases to carry out the function for which it was designed. Clinically this failure is expressed for the left ventricle by disabling dyspnœa and progressive effort intolerance. For the right ventricle a persistent elevation of right atrial pressure produces the effects of systemic venous hypertension, as evidenced by fluid retention and ankle œdema.

It must be stated without reservation that the heart was designed to maintain an adequate supply of oxygen to the tissues. This oxygen supply is the final yardstick by which all the metabolic processes are measured. Between an unlimited supply of this element in the external atmosphere and the body's metabolic pool, lie variable and complex parameters of cardio-vascular function—the most important of which is the cardiac output. The output of the heart can be regarded as a good index of the efficiency of the pump.

To revert to simple mechanical analogies, what features are responsible for the breakdown of a pump? Briefly they are two: either (i) the pump is worked to excess and asked to perform under systems of loading for which it was not designed, or (ii) a spontaneous and unpredictable breakdown of a component part may occur. The same events may happen to the human heart. Firstly, as the result of a prolonged and frequently preventible pathological process, an excess or unphysiological load may be encountered with which the myocardium cannot contend. Secondly, by chance or by disease, a cusp may rupture and the physical integrity of the whole is jeopardized by a single component part.

The most important single component of the heart is the myocardium. Not only must it respond to unphysiological loading by an adequate cardiac output, but it must at the same time maintain an efficient intracellular energy transformation system. Once this metabolic chain is weakened, breakdown becomes inevitable. For those of you who are interested in detailed cardiac physiology, the introduction of that complex concept of myocardial contractility is at once apparent.

The following is by far the most important theme of this paper. The heart is subjected to two varieties of loads. It is asked to pump either: (i) volume loads or (ii) pressure loads.

[1] Read at a plenary session on heart failure, Australasian Medical Congress (B.M.A.), Tenth Session, Hobart, March 1 to 7, 1958.

What is meant by volume loads? Simply high stroke volumes ejected against low vascular resistances or pressure gradients. By contrast, pressure loads are those in which a normal or reduced stroke volume is ejected into high resistance pressure channels.

One of the complexities of cardiac failure lies in the fact that the two principal pumping chambers are designed to attain their greatest efficiency under widely divergent circumstances. The left ventricle manages most efficiently with pressure loads, while the right ventricle, because of its anatomical peculiarities, works maximally with volume loads. Conversely, the left ventricle has difficulty adjusting itself to volume changes, while the right ventricle rebels under hydrostatic pressure stress.

Some of the reasons for the great diversity of this problem are now apparent. It is possible to recall instances in which pressure or volume loads are placed either on the entire heart or selectively on either one of the two ventricles. First must be considered the effect of volume loads on the whole heart.

Volume Loads on the Whole Heart.

Volume loads on the whole heart are produced by high output states: (i) thyrotoxicosis, (ii) thiamine deficiency, (iii) arterio-venous aneurysms, (iv) anæmia, (v) pregnancy, and (vi) liver disease. In those instances, when the heart fails the symptoms will be left ventricular in origin, because we know that the left ventricle will fail before the right when stress is produced by increased blood flow. The development of secondary pulmonary hypertension does complicate the clinical picture, because portion of the stress is then transferred to the right ventricle. The clinical recognition of this group of conditions is important, because once the ætiological factor is isolated, complete cure in most instances is possible. Here the circulation has been justly dubbed hyperkinetic. The patient is warm and alert, the pulses are throbbing, the precordium is unusually active, the hands are peculiarly engorged and hot, and blood flows from the unobstructed venipuncture with a bright arterial hue. The cardiac index is usually greater than five litres per minute per square metre determined by either conventional right heart methods or the use of one of the indicator dilution techniques.

Volume Loads on the Left Ventricle.

We come now to the next set of conditions whose volume loads are placed on the left ventricle. These may be set out in two groups: (i) left-to-right intracardiac shunts, due to (*a*) patent ductus arteriosus or (*b*) ventricular septal defect; (ii) valvular insufficiency—(*a*) aortic insufficiency or (*b*) mitral incompetence.

To deal first with the intracardiac shunts: this load is obviously a function of the size of the intracardiac shunt, and will not become significant unless the absolute pulmonary flow is about 20 to 30 litres per minute per square metre—four to six times normal. If pulmonary hypertension develops, which is mainly the end result of the incessant engorgement of the lesser circulation, the volume load is taken off the left ventricle and the dire significance of the burden is transferred to the right ventricle. What was previously a volume load on the left ventricle now becomes a pressure load on the right ventricle. The left-to-right shunt is lessened, but right ventricular function is diminished. Finally a right-to-left shunt may supervene, and the body is faced with the problem of adaptation to the anoxic state.

Insufficiency of Aortic and Mitral Valves.

In aortic insufficiency, the quantity of blood ejected by the left ventricle must equal the normal cardiac output plus the volume which regurgitates back through the aortic valves. The principal cardiac compensation to this burden is a sustained increase in stroke volume sufficient to compensate for the total volume of the regurgitation. To meet this the left ventricle dilates and assumes some of the functional characteristics of the right ventricle. The surface area of this chamber increases markedly, and the degree of myocardial shortening required to eject a particular volume is thereby diminished. In this manner the aortic regurgitation produces the most massive left ventricular dilatation seen in clinical medicine.

The same principle applies in mitral incompetence. With each contraction of the left ventricle there is a bi-directional propulsion of blood, the partition of flow depending upon the relative resistances existing between the left atrium and the aorta. Under such a sustained blood flow, the left ventricle distends to much larger systolic and diastolic dimensions.

Volume Loads on the Right Ventricle.

Mention must now be made of the next variety of cardiac work load—i.e., volume loads on the right ventricle. This situation is one which the heart is capable of handling. The best example occurs in atrial septal defect, in which torrential left-to-right shunts at the atrial level may cause right ventricular stroke volume of upwards of 200 cubic centimetres per beat. How frequently, in the absence of organic tricuspid incompetence, do we see frank congestive cardiac failure under these circumstances? The answer is, rarely. Only when pulmonary hypertension develops—and this is usually a sign of atrial septal defect complicated by abnormal pulmonary vein insertion—do we see this complication.

Pressure Loads on the Left Ventricle.

We come now to consider those situations where there exists pressure loads on the left ventricle. It is common knowledge how excellently the left ventricle continues to beat efficiently with the added load of either significant systemic hypertension or aortic valve obstruction. In these conditions of pressure load the left ventricle responds primarily by hypertrophy of this chamber.

It would seem appropriate at this stage to spend a little time in discussing the two important physiological adaptations caused by an increased demand for ventricular work—ventricular hypertrophy and ventricular dilatation.

Ventricular hypertrophy, as far as the left ventricle is concerned, is a relatively efficient process in terms of energy transformation. It is the main compensation to pressure loads when the myocardial fibres must generate greater tensions to produce the higher intraventricular pressures during systole. Experiences with left heart catheterization have shown that these pressures may reach a figure of between 300 and 400 millimetres of mercury, especially in tight aortic stenosis. In these circumstances the myocardial fibres increase in diameter and the ventricular wall thickens. For the physiologist, the thickened walls have a diminished distensibility and require a greater effective filling pressure to obtain a particular diastolic volume. Myocardial hypertrophy tends to increased utilization of the systolic reserve capacity of the heart with some sacrifice of distensibility. This is an exaggeration of the normal response of the adult heart to increased physiological loads. It is not until imbalances of coronary flow occur that myocardial contractility diminishes and the ventricle begins to dilate and fail.

Mention has already been made of the problem of ventricular dilatation. Through dilatation an increased stroke volume is attained without a corresponding increase in myocardial shortening. Ventricular dilatation therefore uses the diastolic reserve volume for increasing stroke volume and is a pathological process. It is the right ventricle which employs this method of adaptation.

Pressure Loads on the Right Ventricle.

The next group is the most important to be considered in this discussion of cardiac failure—that in which the morbid process or its secondary effects have placed a pressure load on the right ventricle. The conditions may be stated as follows: (i) pulmonary hypertension: (*a*) primary, (*b*) secondary to mitral valve obstruction, left ventricular failure or pulmonary disease; (ii) pulmonary valve obstruction—pulmonary stenosis. Of these

abnormalities, mitral valve obstruction and pulmonary stenosis are surgically correctable.

A feature common to all the above-mentioned conditions is that the effective forward cardiac output is normal or diminished. Life is maintained because the systemic blood flow is partitioned by a process of selective vasoconstriction. The venous oxygen reservoir is invaded, and the functional integrity of the body as a whole is maintained at the expense of individual organ function. Derangement of renal electrolyte excretion is an expression of this life-sparing process which has far-reaching effects.

It has been customary in the past to refer to this as low-output cardiac failure. I think that one of the most significant advances which have been made in our recent knowledge of cardiac failure is the discovery that the term "low" applies only to the forward ejection of blood. As the right ventricle hypertrophies, this reaction in combination with its normal dilated state causes the tricuspid valve to become more incompetent. With increasing pressure loads, more and more blood is propelled in a retrograde fashion. The effective forward output then becomes the difference between the total right ventricular stroke volume and the regurgitant flow.

The magnitude of the back flow through the functionally incompetent tricuspid valve can be considerable, being partly determined by the relative sizes of the two outlets of the right ventricle—the tricuspid and pulmonary valve orifice area. Another determinant is the difference between the mean right atrial and mean pulmonary artery pressures. This bi-directional output of the right ventricle helps to produce the final breakdown, because when the ventricle fails, it does so in a functionally high output state.

Why does not the same mechanism apply to the left ventricle? The answer is to be found in the anatomical manner in which the mitral valve leaflets are inserted into the body of the ventricle.

Fluid Retention and Œdema Formation.

Another controversial field must be entered. For almost a decade discussion has ranged almost to the point of acrimony about what was the pathogenisis of fluid retention and œdema formation in cardiac decompensation. Was it essentially a hydrostatic effect from increased venous pressure, or were primary renal adjustments to blame? The years have not resolved the backward and forward failure controversy, but if it has done nothing else, time has demonstrated what an arbitrary subdivision it is.

The mechanism of many of the primary salt-retaining reflexes has been clarified. Complex interrelationships involving receptors in the pituitary-adrenal axis are involved, and now we know that the final retention of sodium is a renal tubular effect via the potent mineralocorticosteroid-aldosterone.

It is also known that there are volume receptors situated in the lungs, the left atrium and the great veins. It will not be long before the precise interrelationship of all these factors, and how extracellular osmolarity is maintained, are known.

But at times it is possible to seek refuge in simpler teleological concepts. No matter what the mechanism of salt and water retention is, right venricular failure of the congestive type is really a high-stroke output failure. If a pump is to deliver large stroke volumes, it must be primed with large amounts of fluid. If the degree of tricuspid incompetence is not a fixed reflux and varies from minute to minute with exercise, respiration and emotion, then there must exist a suitable upstream reservoir to allow adequate dynamic resiliency. This, in the human subject, is the caval system. It would seem that one of the reasons for fluid retention is a little more apparent.

All the aspects of this large problem have not been covered. It has been impossible to deal with those conditions in which unusual constriction and limitation are placed on ventricular contraction — especially cardiac tamponade and constrictive pericarditis. In these conditions the diastolic reserve of the heart is limited, and compensation must proceed along the lines of tachycardia and increased systolic reserve output.

Pulmonary Heart Disease.

Time allows only of mention of the pulmonary forms of heart disease—especially anoxic cor pulmonale. As the result of intrinsic lung disease, the total pulmonary oxygen diffusion surface is diminished, and oxygen transport can be augmented only by an increased alveolo-capillary oxygen gradient. This requirement is satisfied by man's living in a permanent cyanotic state.

Conclusion.

In conclusion, let it be said that "the subject of cardiac failure is not an easy one—the horizons are as distant as before". However, if we approach our clinical problems with an appreciation of the theoretical background, possessing the modern tools which we have to make a definite diagnosis, then our knowledge must improve. It has become my experience over the years that no heart is too sick for the cause of its infirmity to be sought, and no patient should be labelled with that stigma of clinical indifference: "Oh! Just another cardiac."

Summary.

1. The human heart fails because of a loss of myocardial contractility.

2. This loss of contractility follows either (i) defective coronary blood supply or (ii) fatigue of the actomyosin-adenosinetriphosphate energy system following long-sustained and unphysiological loads.

3. Unphysiological loads are either volume or pressure loads.

4. The left ventricle fails more rapidly under volume loads, the right under pressure loads.

5. High-output cardiac failure is mainly left ventricular failure under volume loads.

6. Low-output cardiac failure is characterized by (i) low effective stroke volume, but high total right ventricular stroke volumes, (ii) increasing tricuspid incompetence, (iii) marked salt and water retention.

7. Pulmonary heart failure is of either the anoxic or the pulmonary hypertensive variety.

THE MANAGEMENT OF HEART FAILURE.[1]

By J. H. HALLIDAY,
Sydney.

IN his remarks on the mechanism and physiological aspects of heart failure, Dr. Bruce Sinclair-Smith has summarized the present knowledge of this complex and as yet incompletely understood march of events. Whether the failure is acute or chronic, and whether it is associated with high or low cardiac output, the heart is unable to meet the demands of the tissues for oxygen and other nutrients. The chief clinical manifestations are due in most instances to abnormal retention in the body of salt and water. Salt retention is the result of diminished renal blood flow and associated endocrine disturbances with a consequent retention of water. Fluid accumulates not only in the extracellular compartment of the body, but also intravascularly, leading to increased blood volume or hypervolæmia.

Definitive Diagnosis.

It is, of course, essential to determine initially that the patient's signs and symptoms are due to heart failure.

[1] Read at a plenary session on heart failure, Australasian Medical Congress (B.M.A.), Tenth Session, Hobart, March 1 to 7, 1958.

A great deal has been added to our clinical armamentarium in recent years to assist us in reaching this decision at the bedside. An example of this is the study of the jugular venous pulse, admittedly not easy to master, but rewarding beyond measure. Analysis of its height, form and timing yields most valuable evidence of the presence of failure as such, tricuspid incompetence, pulmonary hypertension and constrictive pericarditis.

Once it has been decided that the heart is in failure, two further questions must be answered, and the correct answers are by no means always easy to obtain. The first of these two questions involves the determination of the exact nature of the underlying cardiac disease. This may be a real problem in acute failure with rapid heart rates, when abnormal physical signs in the heart may become masked or even muted until the failure is controlled. Apart from this difficulty, the final answer in some cases requires complex laboratory investigations as mentioned by Dr. Sinclair-Smith, especially when surgical intervention is being considered. The second question to be answered is why heart failure has developed. Precipitating factors, which are present in about half of all cases of failure, may arise within or without the heart. Examples of the former are the onset of an arrhythmia with rapid ventricular rate, acute rheumatic or other type of myocarditis, and myocardial infarction perhaps without significant chest pain. In the important group of precipitating causes extrinsic to the heart are excessive physical exertion and emotion, systemic infections (particularly respiratory), pulmonary infarction (again often without the usual clinical manifestations), thyrotoxicosis, severe anæmia, thiamin deficiency, surgery, anæsthesia and blood loss. With the correction or control of these, failure may disappear or may respond more favourably to other measures. The role of repeated pulmonary infarctions in severe or chronic failure with low cardiac output should be emphasized, as appropriate anticoagulant therapy may contribute significantly to the patient's recovery.

The importance of a full and precise diagnosis is all the more necessary today with the rapidly expanding scope of reparative and curative surgical procedures, some of which, to the older of us, have something of a sputnik aura. While it is true that surgery should if possible be undertaken before the onset of failure, brilliant results may at times still be obtained surgically in certain conditions after medical control of an episode of failure. An example is successful valvotomy in mitral stenosis with severe pulmonary hypertension, associated with right ventricular failure and tricuspid incompetence.

Management.

The importance of the three main factors usually responsible for heart failure—to wit, myocardial insufficiency with inadequate cardiac output, diminished renal blood flow and pituitary-adrenocortical imbalance—varies with the stage of the failure. Inadequate cardiac output is of primary importance and determines the degree of disturbance of the other two factors.

For the purposes of this discussion heart failure may be divided into the following four stages of progressive severity.

1. In the early mild phase, myocardial insufficiency alone is largely responsible for the clinical features. Treatment consists in reducing the work load on the heart by rest, both physical and mental, weight reduction when appropriate, and digitalization.

2. In the second stage the measures just mentioned are not adequate, and the consequences of diminished renal blood flow—sodium and water retention—become apparent. Restriction of sodium intake is now also required.

3. With further deterioration in cardiac output, renal blood flow becomes disproportionately even more diminished, and the combination of rest, administration of digitalis, and salt restriction no longer controls the failure. Properly administered diuretics, of which the mercurial compounds given parenterally are by far the most effective, should now be employed in addition.

4. The final or irreversible stage is reached when the failure proves refractory to all forms of treatment, renal blood flow is fractional, and bizarre electrolyte disturbances, retention of water in excess of that of sodium, and evidence of renal and other organ dysfunction appear and progress to a fatal outcome.

These stages merge into each other, and it must always be borne in mind that any sudden deterioration or unexpected regresson of the patient's condition demands a meticulous scrutiny of every detail of treatment and a complete review of the over-all clinical picture, especially as regards the precipitating causes of failure just mentioned. Therapeutic measures, when not carefully and constantly watched, may in fact be responsible for the downward march of a patient from the third to the fourth stage of failure.

It follows from this admittedly over-simplified classification that, in the more severe degrees of failure, not only are our therapeutic resources more severely taxed, but also they have to be used with the greatest care and with constant observation of the patient's progress.

The remaining time will be devoted to comments on digitalis, salt and water retention and the use of diuretics.

Digitalis.

Since its rediscovery by Withering in 1785, this unique drug has remained entrenched as the most important agent in the treatment of heart failure. Its chief action is to increase the systolic force of the myocardium, thereby raising the cardiac output, reducing the end diastolic pressure in the ventricles and lowering the venous pressure. There is no contraindication to its use in failure, and except in rare instances the drug should be given by mouth. It is most desirable that a practitioner should use not more than two or three of the multitude of digitalis preparations available, as otherwise he cannot acquire an adequate knowledge of the proper administration of the drug. Digitalis leaf, digoxin and digitoxin cover most situations encountered in clinical practice. No specific digitalizing or maintenance dosage exists, and the results of mild overdosage in the maintenance stage may easily be missed. Toxic manifestations, due either to the patient's unusual susceptibility or to unrealized previous ingestion of the drug, may follow the administration of large doses, quickly repeated for the purpose of rapid digitalization. More dangerous is toxicity arising in severe protracted cases of heart failure. The patient may have been maintained on the same dosage for some time, and then some intercurrent factor produces profound biochemical disturbances with depletion of potassium. This renders the heart more sensitive to digitalis, and the resulting anorexia, nausea and vomiting may not be recognized as being due to digitalis, but ascribed to the heart failure itself. Digitalis administration should be suspended temporarily, and either potassium chloride or potassium citrate should be given in doses of three to six grammes daily by mouth, or if necessary intravenously.

If the patient's condition appears to warrant intravenous injection, it must be established beyond any doubt that no form of digitalis has been taken for four weeks previously. Even then only half the estimated digitalizing dose should be given, slowly and well diluted, and the patient observed for at least an hour subsequently. It may then be possible to continue with oral administration; otherwise half the initial dose may be given intravenously after two hours, and if necessary once more after the same interval.

Salt and Water Retention.

Retention of sodium becomes of importance in the management of the second stage of heart failure, and the degree of deficiency of sodium excretion varies directly with the severity of the failure. Thus the patient with mild failure may be capable of taking four to eight grammes of sodium chloride daily without significant sodium retention, and the patient with moderate failure two to three grammes, whereas many patients in severe failure can avoid further sodium retention only if the daily intake of salt is limited to 0·5 to 1·0 gramme (200 to 400 milligrammes of sodium). Thus the degree of salt restriction advised should be tailored to the severity of the failure. Heart failure persisting in a patient should not be regarded as refractory until it proves resistant to treatment which includes a sodium intake of 200 milligrammes a day—a

regime certainly not easily achieved or tolerated. It is doubtful whether there are any other primary electrolyte disturbances associated with uncomplicated heart failure, and restriction of salt intake alone is rarely responsible for untoward effects. However, when mercurial diuretics are administered in conjunction with a very low sodium intake, the situation is very different, and without constant observation extremely serious effects may arise. Some reference to these will be made subsequently. As has already been stated, retention of water in the body is secondary to that of sodium, at least until failure approaches the refractory phase. Accordingly, in the milder degrees, the patient's fluid intake need not be restricted. However, when control of the failure is difficult to attain, fluid intake should be limited to about 2000 cubic centimetres a day. It is interesting to observe that not infrequently this is all that the patient desires. Some patients in severe and protracted failure retain water in excess of sodium—so-called water intoxication. In these circumstances fluid intake should be restricted further, and for a time should be only 500 cubic centimetres in excess of the preceding 24-hour urinary output.

Diuretics.

It should now be clear that a diuretic response in the less severe forms of failure will follow improved myocardial function, either as a result of rest and administration of digitalis, or with other appropriate measures such as the giving of thiamine or anti-thyroid drugs and the correction of anæmia. If such measures are inadequate, diuretics should be given either by mouth or by injection. It must suffice to say that diuretics for oral administration, prior to the recent introduction of chlorothiazide, have been of value only in the milder degrees of failure. An effective diuretic for oral use will represent a major advance in the treatment of heart failure, and present reports suggest that chlorothiazide will prove to be a safe and potent oral diuretic. This compound, in contrast to other carbonic anhydrase inhibitors, causes an almost equivalent excretion of sodium and chloride without the production of a metabolic acidosis. It has proved effective in the treatment of patients who had previously responded poorly to mersalyl, and has also been used in conjunction with mersalyl with good effect. The dosage is of the order of one gramme given twice a day, and diuresis usually follows after two hours. In mild cases 0·5 to 1·5 grammes given on three or four days a week may be sufficient.

The organic mercurial diuretics have been used for many years, and remain the sheet anchor of treatment for the removal of œdema fluid. Perhaps familiarity has been responsible for the tendency for them to be prescribed at standardized intervals rather than according to the needs of the patient at the time. This is not a good practice, particularly in the more severe types of failure, in which it may lead to serious sequelæ.

It is a curious fact that the organic mercurial diuretics are not as generally used in left ventricular failure, in which they are equally effective, as in the types associated with systemic congestion and peripheral œdema. The chief indications for their use may be summarized as follows: (i) congestive failure not relieved by rest, digitalis or sodium restriction; (ii) congestive failure, severe from the onset; (iii) left ventricular failure recurring despite full digitalization and sodium restriction; (iv) recurrence of symptoms with an established critical weight level; (v) inability or unwillingness of the patient to adhere to a diet of low sodium content.

As is well known, there is little direct correlation between the amount of œdema present and the severity of the failure. For all patients, especially the aged and debilitated, the initial dose should not exceed 0·5 cubic centimetre given subcutaneously or intramuscularly, but not intravenously. The optimal dose is the smallest which produces satisfactory diuresis. A massive response carries a definite risk of over-depletion of sodium, with the development of a severe shock state. Rarely should mercurial diuretics be administered daily, because of the real danger of producing severe electrolyte disturbances. The interval between injections should be determined after proper consideration of the patient's condition and the response to the previous injection. When a patient fails to show a satisfactory response to a previously effective dose of, say, two cubic centimetres, careful stock should be taken of the situation and, if possible, the serum electrolyte pattern ascertained. This may reveal an excessive diminution of chloride, associated with some degree of alkalosis. The administration of ammonium chloride in divided doses of four to six grammes daily for two or three days before the next injection of the mercurial will usually be followed by satisfactory diuresis. However, ammonium chloride is contraindicated in the presence of renal or hepatic insufficiency, or when a state of metabolic or respiratory acidosis already exists.

In the more resistant types of failure, lack of response to a previously effective dose of the mercurial compound may indicate a more serious state of affairs, due to an intercurrent complication, to inexorable progression of the failure, or to ill-advised repeated administration of the mercurial in association with rigid salt restriction. This is where the third and fourth stages merge, and the implications are of the gravest. Electrolyte patterns show complicated disturbances, and there are variable degrees of renal failure and symptom complexes due to gastro-intestinal, cerebral or renal dysfunction. Discussion of this difficult sphere of diagnosis and management will be confined to the so-called low salt syndrome or hyponatræmia. Serum sodium levels of the order of 100 to 130 milliequivalents per litre (normal, 140 to 143 milliequivalents per litre) are not infrequently met with in the very persistent or refractory stages of failure. In only a small fraction of such cases is actual sodium depletion responsible and the administration of salt indicated. This is precisely the situation mentioned earlier, when a comprehensive review of the patient's condition, symptomatology and treatment is essential.

It is not possible here to elaborate this theme beyond saying that the clinical picture of sodium depletion is one of acute onset and progression following such happenings as quickly repeated massive diuresis or paracenteses or severe gastro-intestinal disturbances. On the other hand, hyponatræmia associated with sodium dilution presents with a more gradual onset of symptoms and deterioration, and results from intercurrent complications (perhaps unrecognized), progression of the failure or errors in management. This situation may be further complicated by the presence of intrinsic renal or hepatic disease. In both types of hyponatræmia mentioned, many of the clinical features are non-specific and are very similar to those due to other disturbances or complications of heart failure, such as pulmonary or cardiac infarction, digitalis intoxication or renal failure.

Treatment obviously will vary with the cause of the low-sodium state. If it is thought to be due to sodium depletion, sodium should be given by mouth if possible, or otherwise intravenously—300 cubic centimetres of a 5% solution of sodium chloride. This should be given slowly, and the patient should be carefully observed for some hours before the injection is repeated. If sodium dilution is believed responsible, fluid intake must be restricted to a minimum, the question of digitalis toxicity reviewed and potassium chloride given by mouth or intravenously. The value of steroid therapy or alcohol in this condition is not as yet clearly established, but a trial may be warranted.

Conclusion.

It is regretted that it has not been possible to discuss such important matters as the role of anti-hypertensive medication in hypertensive cardiac failure, the place of oxygen, the value of acupuncture or Southey's tubes in neglected patients with gross anasarca, or the use of exchange resins, or to consider the management of cor pulmonale or left ventricular failure.

However, it is hoped that the limited presentation of some of the problems of treatment and of its application will have served to indicate that, while there has been considerable improvement in recent years in our capacity to help patients with failing hearts, greater care and observation are now required, not only to ensure the best possible results from treatment, but also to avoid adding any further load to the already distressed pump.

THE ROLE OF THE SURGEON IN HEART FAILURE.[1]

By IAN MONK,
Sydney.

THE following remarks are made from the point of view of the surgeon, so that the first observation I would make is that in a certain proportion of cases of heart failure the underlying cause is mechanical. Therefore the diagnosis "cardiac failure" is inadequate unless the cause is specified, because in certain cases the relief of the mechanical derangement by surgical means may cure the patient. The second generalization one should make is to point out that it may be too late to ask the surgeon to operate on the heart by the time it has failed, because if any organ is subjected to sufficient stress for long enough there comes a time when irreversible changes occur.

Whatever one may adopt as a definition of heart failure, it is essenial to realize that in certain cases physiological tests will reveal serious disturbances long before the classical symptoms and signs indicate that established cardiac failure is present. Although pleas for early diagnosis are made to the point of tedium, the correct timing for any surgical intervention is one of the secrets of success. This is a responsibility which always must be shared by the medical profession as a whole.

There are three fundamental types of heart operation: (*a*) the relief of an obstruction; (*b*) the closure of a fistula; (*c*) indirect operations. Under these three headings surgical relief can be offered as follows:

The relief of an obstruction:

1. Constrictive pericarditis.
2. Tricuspid stenosis.
3. Infundibular and pulmonary stenosis.
4. Mitral stenosis.
5. Aortic stenosis.
6. Coarctation of the aorta.

Closure of a fistula:

1. Patent ductus arteriosus.
2. Arterio-venous fistulæ.
3. Atrial septal defects.
4. Ventricular septal defects.
5. Anomalous pulmonary venous drainage.
6. Rupture of the sinus of Valsalva.

Indirect operations:

1. The correction of inadequate pulmonary blood flow by the anastomosis of a systemic artery to a pulmonary artery.
2. Reduction of pulmonary congestion by anastomosing a pulmonary vein to the azygos vein.
3. Reduction of cardiac backflow in tricuspid incompetence by ligation of the inferior vena cava.

The remarks which follow will be confined to the more common cardiac lesions.

Obstructive Lesions.

Mitral Stenosis.

In the last ten years, the operation of mitral valvotomy has brought relief to many thousands of patients suffering from mitral stenosis. In favourable cases the operative risk should not exceed 1%, and about two-thirds of all the patients treated surgically obtain a good result (Wood, 1954).

Broadly speaking, operation is indicated if the degree of stenosis of the mitral valve causes cardiac embarrassment. When the patient's symptoms are slight it may be difficult to decide whether operation should be performed, so that cardiac catheterization may be necessary to determine the severity of the valve obstruction. In the vast majority of cases, however, there is no difficulty in deciding the question of operation on clinical grounds.

In advanced or neglected mitral valve disease, when gross cardiac failure is present, the clinical features become obscured by such phenomena as functional dilatation and incompetence of the mitral and tricuspid valves. The physiological data obtained by cardiac catheterization will reveal the true state of affairs in most cases, but occasionally exploratory cardiotomy may be necessary to determine the exact diagnosis (Brock, 1956).

Operation is best avoided if possible during adolescence or in young adults, because of the risk of reactivation of rheumatic carditis. Pregnancy, on the other hand, may bring to light previously unsuspected mitral stenosis and may actually be an indication for operation. Although previous embolism associated with mitral stenosis is an indication for operation, it may increase the surgical risk, as does long-standing pulmonary hypertension, heavy calcification of the mitral valve or any evidence of mitral or aortic incompetence.

Aortic Stenosis.

For anatomical and pathological reasons, aortic valvotomy is a less satisfactory operation than mitral valvotomy. However, the operation of transventricular aortic valvotomy gives reasonably good results in the majority of cases, with an acceptable mortality, providing that it is performed early enough (Brock, 1957). The most important fact concerning aortic stenosis that has emerged in recent years is that early symptoms of cardiac stress, or signs of left ventricular strain in the electrocardiogram, should lead to an investigation of the left ventricular pressures. If a high-pressure gradient between the left ventricle and the aorta exists at the aortic valve, then operation is indicated (Brock, 1956). Ultimately, when open heart surgery becomes safe, it is likely that the results will be improved for this condition.

Obstruction to the Right Ventricle.

Obstruction to the outflow tract of the right ventricle may occur as an isolated lesion, such as pulmonary valve stenosis, or a combination of abnormalities may be seen as in the tetralogy of Fallot.

Pulmonary valve stenosis, either alone or accompanied by other abnormalities, is a relatively common lesion. Final assessment of this condition must depend on cardiac catheterization, and frequently the indications for operation depend on the degree of obstruction present, as evidenced by the pressure gradient across the pulmonary valve.

In 1948, Brock adopted a policy of pulmonary valvotomy for this condition in place of the indirect operations then popular (Blalock, 1946). This operation is performed through the right ventricle. Brock was able to show, first, that the heart tolerated this interference surprisingly well, and he was able to produce results as good as, if not better, than those following indirect operations except in the presence of heart failure, when the risks become very high. However, some surgeons have felt dissatisfaction at their results, because follow-up studies have shown that a serious degree of obstruction may still remain, despite an apparently successful operation. The general trend at the moment is to favour a direct operation on the pulmonary valve, by opening the pulmonary artery with the aid of either hypothermia or the cardiac by-pass.

Constrictive Pericarditis.

Although this condition is comparatively rare in this country, when it does occur the diagnosis is apt to be unduly delayed. In the past the results of operation have been somewhat unpredictable because of this delay in diagnosis, and also because of inadequate freeing of the heart from its constricting membrane. When adequate treatment is carried out early for this condition, the risks of operation are small and the results are good, and there is now general acceptance of the principle that the heart should be freed from its constriction on both sides, including the right atrium and entrance of the venæ cavæ.

[1] Read at a plenary session on heart failure, Australasian Medical Congress (B.M.A.), Tenth Session, Hobart, March 1 to 7, 1958.

Other Obstructive Lesions.

Infundibular stenosis may occasionally occur as an isolated obstruction to the outflow tract of the right ventricle. Brock has produced convincing evidence that in his hands his operation of infundibular resection produces good results (Campbell *et alii*, 1954). However, at the present time this problem is bound up with the whole question of open heart surgery.

Tricuspid stenosis and *subaortic stenosis* are rare obstructive lesions, for which surgical relief can be offered (Brock *et alii*, 1956). Coarctation of the aorta will be referred to later in connexion with heart failure in infancy.

Fistulous Communications.

Patent Ductus Arteriosus.

All patients with this condition should be operated on unless very unusual circumstances are present, because the operative mortality rate in competent hands is a fraction of 1%.

Very occasionally severe pulmonary hypertension may complicate this lesion. If this is of such a degree that the pressure in the pulmonary artery equals, or occasionally exceeds, that of the systemic pressure, general opinion at the moment would suggest that such a condition is inoperable. In practice, a final decision may have to be left to the findings at exploratory thoracotomy. If temporary occlusion of the ductus shows that the pressure in the pulmonary artery can be kept below the systemic blood pressure, then ligation or division of the ductus should be carried out.

Septal Defects.

These developmental abnormalities may occur singly or in association with other defects. Such mechanical derangements often cause gross disturbances of hæmodynamics, and the assessment of such patients is a complex physiological matter. However, in the last year or two thoughts have become clearer on this subject.

If a patient with an atrial septal defect presents with minimal symptoms, but investigations show that the pulmonary blood flow is at least two and a half times greater than the systemic blood flow, then operation should be recommended. Under hypothermia the circulation can be stopped for about ten minutes and the defect closed under direct vision. This can be accomplished with a very low surgical mortality (Bedford, 1957). However, if the defect is an abnormality of the primum type, or if high fixed pulmonary hypertension or heart failure is present, then the condition is probably inoperable by this method.

Patients with ventricular septal defects, on the other hand, are much more prone to develop pulmonary hypertension and heart failure. For this reason, and because a prolonged period of open heart operating may be necessary to effect closure, the cardiac by-pass is necessary if such a defect is to be treated surgically.

Other Fistulous Communications.

Extensive experience in three major wars has shown that traumatic lesions in the periphery which cause arterio-venous communications result ultimately in heart failure. All such lesions should be corrected surgically.

Other rare lesions which have been successfully treated surgically are rupture of the sinus of Valsalva and aortico-pulmonary fistula.

Indirect Operations.

Indirect operations are those which bring relief to a patient without correcting the underlying abnormality. The brilliant pioneer work of Blalock and Taussig in providing a remedy for cyanotic heart disease is now a matter of surgical history (Blalock, 1946). They recognized that the cyanosis and disability were caused in many cases by the passage of far too little blood to the lungs, and the remedy devised was to anastomose a systemic to a pulmonary artery, so that inadequately oxygenated blood was conducted to the lungs. The immediate success of this operation the world over ushered in a new era in cardiac surgery.

Another indirect operation was that devised by Sweet for the relief of pulmonary hypertension associated with mitral stenosis. He anastomosed the azygos vein to the apical lower lobe pulmonary vein. The symptomatic relief brought to a small series of patients was quite striking; but about the same time the operation of mitral valvotomy became established and this operation was abandoned (Sweet, 1957).

Another indirect operation which occasionally may still have some application is that of inferior vena caval ligation (*Cassio et alii, 1949*). In certain types of heart failure the tricuspid valve becomes incompetent, so that the blood put out from the heart at each beat is partly expelled back. This causes dilatation of the cervical veins, ascites, œdema of the lower limbs and an enlarged, pulsating liver. Under these circumstances the effect of ligation of the inferior vena cava on the disordered physiology is to prevent the blood that flows back through the incompetent tricuspid valve from being driven into the large venous reservoir of the lower half of the body.

In a very small personal experience, one patient was able to lead a socially useful life for over two years, having previously been virtually bedridden for the previous three years.

Special Considerations.

Heart Failure in Infancy.

In recent years, attention has been drawn to the fact that a certain number of young infants with lesions such as patent ductus arteriosus succumbed from heart failure. At this age the diagnosis of patent ductus, coarctation of the aorta or atrial septal defect may be very difficult by any means. These are surgically remediable lesions, and under certain circumstances there may be enough doubt to warrant an exploratory thoracotomy. No infant should be allowed to die of heart failure until a surgically remediable lesion has been excluded.

Cyanotic Heart Disease.

Special mention is made of such conditions as Fallot's tetralogy, because the general trend everywhere is to favour direct operations on the heart, with the aid of either hypothermia or the cardiac by-pass. At the moment of preparation of this paper, cardiologists are avoiding referring patients for operation until the position becomes clarified in relation to open heart surgery. However, when the condition of a small child with Fallot's tetralogy is obviously deteriorating, there should be no hesitation in submitting such a patient to an anastomotic operation. This is a well-tried, well-proved operation without great risk.

Open Heart Surgery.

Open heart surgery is now an established procedure. It remains to be seen whether hypothermia or the cardiac by-pass or both methods will remain in use for cardiac surgery.

Cooling the body down to 29° or 30° C. enables the circulation to be stopped safely for 10 to 12 minutes. Atrial septal defects, pulmonary stenosis and aortic stenosis are examples of lesions which have been corrected under direct vision by the use of this method. Although the surgical manipulations within the heart under hypothermia are limited mainly by the short time available, current investigations in certain centres suggest that this period may be prolonged.

A cardiac by-pass, or so-called artificial heart-lung machine, has been used successfully now on many hundreds of patients. Variations and improvements in technique are reported almost weekly, and although many problems remain to be solved, it is likely that in the near future most operations on the heart will be performed with the use of such apparatus. It is possible, also, that an

artificial circulation may be used as a method of resuscitation.

Conclusions.

1. Certain cardiac lesions of a mechanical nature can be relieved by operation, so that every patient presenting with heart failure should be viewed with this in mind.

2. In a patient with a surgically remediable cardiac lesion, the classical symptoms and signs of heart failure sometimes indicate the imminence of death rather than the necessity for operation.

3. When operations can be performed on the open heart for prolonged periods safely, many techniques in use at present will be improved and the scope of surgery will be widened.

4. Even when the cardiac by-pass finds safe general application, many problems will remain unsolved by the mechanical manipulations with which surgery is mostly concerned at present.

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THE FUTURE OF THE AUSTRALIAN ABORIGINAL.

By B. B. BARRACK,
Brisbane.

THE great majority of our aborigines, both full blood and those of mixed blood, live in Australia, north of the Tropic of Capricorn. In considering the future of the aborigines of Australia, there is also to be considered the development of this vast northern area, from our own Australian point of view; if we fail to develop it, it is more than likely others will, or others might use it to secure a foothold on this continent before infiltrating into the more temperate southern zones.

It does not seem possible that a pure European people will ever find a permanent home in these regions; the climate with its extreme dry, followed by a severe, shorter, wet season, with excessive heat and humidity, is such that only a limited number go there to seek their fortune. There will always be the adventurous ones; there will be some seeking to evade their responsibilities; there will be those evading the law; but whatever their reason for going there, there will not be enough of them, and there will not be the desire to form permanent settlements except in isolated cases—if it is not suitable for a white man, it is even less suitable for a white woman. It has been clearly shown in the past that Europeans can live there, that the climate, if hot, is not unhealthy, and there are few diseases to make life more difficult or hazardous; but in spite of that, in spite of modern communications and modern amenities, very few have any desire to settle and bring up families there. Immigrants from Europe go there mostly to make sufficient money to start in business in the southern cities, and again few are permanent settlers, and the same applies to those who seek work in the mines.

Since the end of the last war there has been considerable development in Darwin, where the population now is in the vicinity of 10,000, and also in all the other settlements. Air travel, improvement in roads, particularly the bitumen highways from Alice Springs and Mt. Isa, mining and to a lesser extent experiments in agriculture have all contributed to this development, but in spite of this it is not likely to make a great deal of difference in so vast an area. The pastoral industry is the only big industry in the whole of the area from Cape York Peninsula to the Kimberleys, and is likely to remain so; large areas have been taken up and developed now for many years, and great improvement has been made recently, so that people are no longer isolated as they were in the past. Air travel, radio, refrigeration and the amenities that go with modern life have made life on these stations safer and more livable. In this industry great help has been obtained in the past from our aborigines, and in the future they and their descendants could be a permanent source of help to work and maintain this industry.

The vast majority of Australians are hardly conscious of the fact that there are still quite a number of aborigines living in this continent. In more recent years the success of several in the arts—Namatjira in landscape painting, Blair in singing and others in motion pictures—have drawn attention to this; in spite of that they are too remote to create but passing interest. It is extremely doubtful if the average Australian could distinguish one from a native of India, a negro, a Melanesian or a Polynesian. The position today is that there are some 75-odd thousand aborigines still in Australia, the majority living in the northern parts, with scattered groups through the deserts down as far as the transcontinental railway, and in these areas full bloods would greatly outnumber the ones of mixed blood. In the south, however, the process of absorption has gone on to the extent that it would be difficult to find a full blood, and in a few generations they will be completely absorbed into the present population.

There is no question that in the past the aborigines have been badly treated. At this point we can look back and see the ill treatment to which they were subjected and feel how unnecessarily cruel and unfair the early settlers in this country were, but I think, given the same circumstances again, we would do very much the same as our forefathers did. It was a case of a nineteenth century culture with its many blessings and its many faults and evils impinging on a stone-age culture of 10 to 15 millennia ago—the oldest stone age culture in existence—a culture of nomads, highly developed but stationary, a culture of hunters with well-defined rules of the tribes in relation to hunting grounds, with very strict tribal laws relating to tribal relationship, leading a communal life with very strict rules of precedence and of relationship in regard to marriage. At the end of the eighteenth and the beginning of the nineteenth century the European had a very definite fear and dread of the primitive coloured peoples of the remote areas of the earth; he had heard reports of the indigenous people of Mexico and their human sacrifices, and he had heard too that many of these primitive peoples practised cannibalism, and so whenever the aborigines of Australia failed to understand the white man's way of life or resisted his intrusion into his tribal hunting grounds, then fear and dread developed into a determination to teach them a lesson, which meant natives were killed; this in time led to further retaliation, with the inevitable elimination of the natives. In some cases friendly relations were established with the aboriginal, and when he was fairly treated and respect was paid to his tribal customs, he became a great help and guide, particularly in exploration in areas where water was hard to find; without him many a white man could not have survived, but the more he helped the white man the more he lost control of his tribal hunting grounds, till eventually he was either exterminated or he was pushed back into the more remote or inhospitable areas. It is only of recent

years that efforts have been undertaken to develop these areas, and in this development he, the native, has played a conspicuous part, but one which is very little appreciated by those he helped.

When the European arrived in this country he found our native Australians different from the inhabitants of all other countries. His colour varied from a deep sooty black to a deep brown. The men particularly were developed along the lines of an athlete as one would expect a nomadic hunter to be; he was well set up, with good shoulders and chest, narrow hips and thin legs. His head showed a receding forehead, accentuated by beetling eyebrows, and broad nostrils suitable to a hot dry climate. He had wavy hair, he grew a luxuriant beard and also a considerable amount of body hair which is unusual in most coloured people. There was, of course, considerable variation in different regions. Quite recently there have been discovered remnants of the Tasmanian negritos who preceded our own natives in Australia, and there was possibly some crossing with them in which the Australian native type predominated. The females were, as compared to the males, more or less nondescript. When young some were quite comely, but like the native women of all hot countries, early childbirth soon reduced any youthful charms to zero. In some full-blooded children, particularly in Central Australia, there is often seen a very definite light colouring of the hair. It has been repeatedly noticed from the earliest times that crossing with the European produced a very rapid lightening of the skin—the half-castes were generally known as yellow boys and girls; this became more evident in the quarter caste, and in the octoroon all colour had disappeared. All authorities that I have questioned are agreed that there is no tendency to throw back in regard to colour, but in some octoroons some broadening of the nostrils persists, and there is also the tendency to retain thin legs. This tendency towards the white in crossing could be due to the fact that the native Australian is of Caucasian origin.

The benefits of Western civilization were in the early days quite useless to these nomadic people, and our diseases and alcohol were a curse—they took a heavy toll. In the first place the diseases of the rest of the world were brought to a people who had no immunity to them; the native could recover from a wound which might easily prove mortal to a European, but he fell an easy victim to measles, influenza, tuberculosis, leprosy and to many of the other diseases with which we have had contact for many centuries. Syphilis, I think, did not play a very important part; he had had yaws before the arrival of the white man, and as yaws and syphilis are so alike and one gives immunity to the other, he was able to stand up to it. Gonorrhœa probably played a part in reducing the birth-rate. Alcohol played a big part in their degradation, and still continues to do so. Apart from active disease, however, there was probably a loss of desire to perpetuate his race. The tribal laws and their culture were handed down by the old men of the tribe in their various ceremonies, and great care was taken to see that their laws and ceremonies were observed to the very letter, but the impact of the newcomers tended to destroy these, and it would seem then that they had no object in life—they were out on a limb. That loss of objective transmitted through the male was an additional factor for their decrease in numbers.

It was estimated that the native population when Cook arrived in Australia was in the vicinity of 300,000; it is now about 75,000, with a tendency recently to stabilization and even a slight increase, but it would not be an increase of the full bloods. This recent stabilization would appear to be a partial adjustment after 170 years to our way of life.

It is of interest to see what happened to indigenous people in other continents when they were confronted with the occupation of their lands by Europeans or by people of more advanced cultures. In the temperate zones of both North and South America, once the newcomers had established themselves by the superiority of their arms, advances were made as more land was required. Their superior arms, their organization, diseases and alcohol all played their part in reducing the native peoples' power of resistance, and with that gone they were gradually absorbed or eliminated, so that today they are represented by some isolated units on reserves, and it is doubtful if there are any full bloods left. In North America they have developed the attitude, and very wisely, that Indian blood is something to be proud of. I think that is something we would do well to consider in the future. In South America in the temperate zone republics descendants of the Spaniards and the Indians are largely employed in the cattle industry; in this environment they seem to have found a way of living to which they are suited and contented. In 1930 the Mestizo (or people with Indian blood in their veins) population of Argentine was about 2%. However, in the central and tropical areas of the Americas it was a different story. Here the native Indians were in such numbers and the Spaniards so few (in spite of the fact that they were the overlords, they did not multiply except in several of the urban centres) that they produced many half-castes, and today the European is gradually being absorbed by the natives to produce perhaps a lighter coloured Indian, in whom the Spanish blood is becoming more and more diluted.

In South Africa the primitive Bushmen and Hottentots were quickly reduced in numbers and pushed back into the more arid regions, so that today few remain; but with the advance of the white man and the opening up of the country the African from the north came in (they were not the original inhabitants), the Dutch also introduced Malays from Indonesia, and later the British brought in Indians to work the sugar fields of Natal. The coloured problem then has practically nothing to do with the original inhabitants; it is due to the late comers after the Dutch had established themselves.

In New Zealand the strong and warlike Maoris put up considerable resistance in the early years of settlement, but eventually they were forced to submit. Disease as usual took its toll, and it looked at one stage that the race would die out. However, since the turn of the century, given considerable concessions and improved medical care, they have adjusted themselves, and of recent years they are increasing. The same applies to the whole of Polynesia; in some cases the natives have been almost completely absorbed as in Hawaii, in other areas they have survived, but in greatly reduced numbers, except in the case of the Samoan and Tongan Islands, where the populations are increasing. In these two groups the missions exert a great influence and alcohol is prohibited.

Coming closer to home, the native Tasmanian negritoes were completely eliminated in just over 70 years.

Apparently the same thing happened in ancient times in Japan, but in that case in reverse, the Proto-Nordic Ainu, the original inhabitants, being absorbed with the exception of a small remnant in northern parts.

In the settlement of Siberia by the Russian, the area was large and sparsely populated. Once the power of the descendants of the Mongols was broken, only moderate resistance was encountered by the groups of Cossacks and traders who were seeking furs. Settlement then took the form of forts and trading posts. There was in the early period a certain amount of fighting, with the result that the number of the natives in these areas was reduced. This went on over a period of several hundred years. A certain amount of cross breeding took place with the natives, but as many of the settlements contained political prisoners including a number of women, there was not the urge to seek the primitive women when others were available. In more recent years the Russian settlers were confined mostly to the areas adjacent to the trans-Siberian railway; more recently still there has been considerable development both industrially and agriculturally, and with the increase in population it would appear that soon there will be only scattered groups of the original inhabitants left in remote areas.

In contrast to the lands inhabited by primitive people, in the densely populated tropical regions of the earth, where there were well-established cultures, in areas unsuited for the normal living of the European in spite of his conquest and the duration of his rule, his stay there has had practically no effect on the indigenous

population. Some cross breeding naturally took place, but it has had no effect on the race and will be eliminated in a few generations.

So it would seem that what has happened in Australia is rather similar to what has happened in other countries inhabitated by more or less primitive people, and it can be said in our favour that, in spite of the fact that our aborigines, with the exception of the Tasmanian, were the most primitive of all these people that I have mentioned, we still have quite a number left after 170 years. This is nothing on which to congratulate ourselves, but it behoves us to try to help those who remain for their good and for our good and for the good of Australia. The remaining natives cannot be confined in an area such as Arnhem Land. If it was possible he would become a museum piece, and in spite of his segregation he would eventually be absorbed or he would die out. He must eventually be incorporated into our civilization, and that means ultimately he will become part of it.

The position today, as has been said before, is that there are some 75,000 native Australians, which includes full bloods and those of mixed blood, still living in Australia. There has been the tendency of more recent years to maintain their numbers, except that the full bloods have decreased at the expense of the mixed bloods. There are still in a few areas such as Arnhem Land, natives in their stone-age state, but these will decrease as they come into contact with civilization. While the old men are still alive they will continue to pass on their laws to the younger generation, but both the young full bloods and those of mixed blood are being educated at missions and schools, so that in another generation or two knowledge of these tribal laws will become less and less as the young ones will have been brought up in the way of life of the white Australian. In spite of that, I believe that there will for a time be a desire varying in different individuals for them to seek the natural outdoor life to which their forefathers were born; legends, like fairy stories to European people, will be handed down which will tend to make them different from their white brothers, until such time as they are completely integrated into the way of the white Australian.

What can be done to help them—the full bloods, the half-castes and the lesser castes? There is not much that can be done for the old full bloods brought up with all his tribal law and beliefs, except to protect them from being exploited. They should be treated with respect for their tribal laws and beliefs, but with a firmness that allows them to retain their self respect, but does not savour of patronage. They are more contented in the environment of the bush on the large cattle stations where great use can be made of them if handled correctly, and it is in this environment that I think their future lies. In the case of the mixed breeds it seems inevitable that we will eventually breed a mixture of white and black Australians, who will be suited to the climate and environment of the tropical north, somewhat like the Spanish-Indian gauchos of the pampas of South America. Considerable thought and experience will be needed to see that he will find a way of life in which he will work and be happy, and at the same time he will be a valuable asset to the country.

The present position is, apart from the town-dwelling natives who will be absorbed more quickly due to their longer and closer contact with whites, that the natives working on stations live much like their forefathers did—under the stars. They get meat and bread from the station and they can supplement this diet by hunting as they have always done. They build gunyahs, but now clothed, this is an unsuitable way of life—they have little or no protection from the weather and if they get wet they are likely to develop respiratory diseases due to their inadequate shelter and wet clothing. If there is a death, they burn the camp to destroy the spirits, so that given huts they could not continue to live where somebody had died. In the future generations this belief will be corrected, but in housing them a special type of hut should be designed suitable for the climate and in which the occupiers could take a pride, in which decontamination of both disease and spirits could be carried on in a way to replace the necessity of destruction by fire. Instead of the straight-sided hut, a hut with an open space between the wall and the roof with an overhang is suggested as more airy, cooler and without giving the suggestion of being hemmed in.

As I have said before, the town-dwelling or near town-dwelling natives will be comparatively quickly absorbed and their colour will lessen in proportion to their mixture with the whites. They will work alongside their white counterparts and in several generations will hardly be noticed. There is not much that can be done to improve their present condition; they are in an environment in which they seem reasonably contented, they associate with the lower strata of white Australians, but some effort should be made to help the children, to give them a better outlook. Education will eventually open up avenues of employment in which they will in time adjust themselves, but it is essential that they should be trained to avoid developing a sense of inferiority. One of the problems that has to be met is the young girl of mixed blood, who is fair game for the youths of her environment. Every means should be taken to see that she has a fair chance in life; if she becomes pregnant, she should receive proper care and everything should be done for the up-bringing of the child. It must be remembered that such children will be the parents of the future in the assimilation of the native Australians into our people.

The natives of the more or less remote areas of North Australia find that work on stations is something that they can undertake, particularly the handling of horses and cattle, an employment to which they are suited and to which they can adjust themselves and enjoy. To work and find enjoyment in your work breeds a contented people, and it is something at which we could all aim. Every effort should be made to see that they are in good hands, to reward them in a suitable way for satisfactory work, to make them interested in their work and to let them vie with each other for seniority and let them know the results of their work. A distinctive type of dress suitable to the climate would help them to take a pride in themselves, and competitive bushmen's carnivals, in which they could display their skill in handling horses and cattle, in station and bush work and in the use of the weapons of their forefathers would serve as a stimulant to improve themselves and add to their enjoyment. In handling them, kindness, with firmness and fairness, is essential as has been mentioned before; the over-sentimental approach is to be avoided, just as cruelty and unfairness are to be avoided. Alcohol is one of their greatest curses—it undermines their health and gives them false courage to defy authority. Soft drinks, known to them as "lolly water", are poor substitutes; a properly brewed hop beer with a very low alcoholic content (2%) could be substituted, and could be sufficiently attractive for them to enjoy it and at the same time keep them reasonably sober. In suggesting such a drink it must be realized that, denied some substitute, they will procure some form of alcohol or drugs which are infinitely worse.

The womenfolk could be employed in station housework, in the store and in cooking. Some in each group could be trained in nursing to aid their fellows, but this would be for future generations, in fact this is an employment particularly suited for some of the young female mixed bloods; that and the care of children would appear an ideal employment for them.

It might be asked, how could such ideas be put into effect? It would be of interest to take a large cattle property that has been in the past and is still in the present successfully worked, almost exclusively, by native Australian labour directed by a white man with the assistance of his wife and one or, at the most, two jackeroos and sometimes without any other white assistants at all. You will find in Arthur Groom's book "Wealth in the Wilderness" a very good description of the workings of such a station in the Kimberley district of Western Australia. Arthur Millard and his wife have been there for 20 years or more, and before him his uncle managed the same property. He has a great knowledge of the country and the natives, he knows the natives probably as well as any other white man in the north, he knows their tribal laws and respects them, and he is firm and kind.

Much could be learnt from men of his type, and young men seeking their future in these parts would do well to serve under men of his calibre, and it would materially help them in directing the future of these native people. His experience can prove that pastoral work of this type can be successfully carried out almost exclusively by directed native labour.

Because we still have some 75,000 native Australians in this country we are in the unique position of being able to profit by our own mistakes of the past and the mistakes of other nations in dealing with a like problem. We are in a unique position because the people we are dealing with can be more easily assimilated than any other native race. We are in a unique position because it lies in our power, if we have the will, to undertake the problem of directing it to the advantage of our natives and of ourselves.

The Commonwealth Department of Native Affairs have done and are still doing good work in the Northern Territory. The same applies to the governments of Queensland and Western Australia, but no over-all policy of direction or coordination exists. A permanent Commonwealth Commission should be set up, free as far as possible from political control, to direct the welfare and education of the aborigines of the whole of Australia, such a commission to consist of three members and to have its headquarters in Darwin, and to consist of one member each from the Northern Territory, Queensland and Western Australia. Their work would be to investigate the work on all the missions and stations, and, if necessary, to obtain the help of such people as they see fit, such as pastoralists, ministers in charge of missions, doctors, dentists and educationalists to aid in the assimilation of our natives—a process which must take time and cannot be hurried. In the constitution of such a commission, no minister of religion should be allowed to be one of the members because of the danger of alienating the support of members of other sects, but great help could be obtained from such missions in their investigations. Much could be learnt by such a commission, mistakes, and there would be many, could be rectified and adjustments could be made, and all for the ultimate good of the natives and ourselves.

In and around settlements and towns where there are natives, people with the welfare of our natives and their descendants at heart could form themselves into committees to try to see if some amenities could be provided for them in their settlements or towns to prevent them drifting to alcohol and crime. Such committees would work somewhat after the principle of Legacy without thought of any gain or advancement except in the satisfaction of improving the lot of our native people and working for the good of Australia.

To summarize, our aim should be for the men and boys to be trained and used in station work, and for the women and girls in domestic duties and nursing, and the establishment of fixed suitable housing for them on the stations where they would become domesticated and educated. This would not come about in one generation; it may take two, three or five, but gradually there would be built up an indigenous population of mixed white and black with their roots in the country and a desire to continue to live there.

It would appear that our natives have adjusted themselves to our civilized diseases, and are now in a better state to combat them, and that, with the help of various hospitals and the use of modern methods and drugs, there is no reason to think that these numbers will now decrease. However, they will definitely tend to decrease if they are not in a contented suitable environment.

What will happen in the future? Future generations will grow up lighter in colour than native Australians and darker than white groups in these areas, who are born in and to the land and who will be the backbone of the pastoral industry in this area. This does not suppose that some will not become land owners in the future. I think that eventually the whole of the north will be populated predominantly by such a mixture of white and native Australians.

The recent move to give full citizenship to our aborigines, both full bloods as well as mixed bloods, is to be deplored. They are at present quite unsuitable for it, and it would be of no value to them—it would give them free access to alcohol and all that goes with it. It has been abundantly proved in the past that free access to alcohol is one of the principal causes of the degradation of primitive people and their eventual extermination. Finally, it is high time that our weekly magazines and periodicals gave up lampooning our aborigines. As long as I can remember we have been regaled with this type of so-called amusing caricature. The same themes have been done over and over again and still they continue. They are in poor taste and often very stupid, appealing only to the lowest mentality, and they have a very definite psychological effect on the natives. We have in the past pushed him down and ignored him, so it is up to us now to do our best to help him regain his confidence in himself and in his offspring, whether they are full bloods or mixed bloods.

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THE COMPENSATION OF INDUSTRIAL INJURIES.[1]

By P. MACDONALD TOW,
Launceston, Tasmania.

NEUROSIS in the industrial worker is a large economic problem. Neurotic illness in industry accounts for a greater loss of working time than do industrial strikes. It also has various unseen effects; for example, it may well affect the worker while on the job as well as when he is away.

Of recent years one of the most disturbing and confusing features in the scene of industrial injuries has been its increasingly complex relation to monetary compensation and organized workers' activities. The original concept of compensation neurosis is nowadays probably far too narrow to encompass the many and strange events which today may follow an industrial accident; or, to put it another way, the variety of ways in which the injured patient may react to his catastrophe. It is so very common now to meet the subject in whom personality, reacting in the context of his trade union and occupational history, completely overshadows other factors in determining the course of events after industrial injury.

Why do men behave like this? Why, in particular, can some men suffer accidents and recover; while others sustain injuries and do not recover from them in anything like the amount of time we should consider normal? What kind of disorder is this characteristic which we call accident proneness? What is the nature of the disease we call compensation neurosis? What is their physiology and their psychology?

Accident Proneness.

For some time now we have invoked the concept of accident proneness in discussing academically the incidence of accidents in injury, and some of you will have heard a good deal about it. I would not like you to assume that in psychological circles we already understand it as a clinical entity. We do not know the nature of the disorder involved, although Dr. Russell Davis, in the Medical

[1] Read at a combined meeting of the Sections of Neurology, Ophthalmology, Orthopædics and Public Health, Australasian Medical Congress (B.M.A.), Tenth Session, Hobart, March 1 to 7, 1958.

Research Council Unit in the psychological laboratory at Cambridge, has done some most interesting work in comparing workers who are accident-prone with those who are not. Using coal miners as subjects, he has shown that under experimental conditions performance in his specially prepared skilled response test was highly correlated with accident history, when this performance was rated on the degree of organization of the discrete responses of which it was composed.

In these experiments Russell Davis compared the behaviour of miners who had sustained more than four accidents with the behaviour of those who had sustained no accidents. The responses of the individuals with a history of recurrent accidents were found to lack that essential quality of skill which lies in the close control of response by stimulus. There are two possible explanations for this finding: either the mechanism, intermediate between receptor and effector by which the response is brought about, is defective; or the mechanism is normal, but is not engaged by the stimulus. Russell Davis remarks that these alternatives may later be found to be compatible, but at present preference of one or other alternative decides the approach for future research. As to the nature of the disorder he says that the activity of the accident-prone man is not entirely at the disposal of the immediate environment in the way that it should be for the efficient performance of a skill.

Though this is the best available work on this subject, clearly it takes us only a certain distance further in our appreciation of these matters in clinical practice. However, it does formulate the matter in scientific language, and records accurately the type of behaviour associated with a history of recurrent accidents. The fact that there is this essential personal ingredient in all accidents brings home to us that what is always important is the player and not the setting of the stage.

Compensation Neurosis.

Let us now turn from the cause to the sequels of injury. Compensation neurosis is a much older concept. The classical post-traumatic syndromes of Professor Alfred Meyer and Sir David Henderson may well appear vague and obscure to the practising general physician, and certainly to many lawyers who have to deal with them in the courts. Nevertheless, these neuroses are well recognized and clearly defined conditions associated with morbid states of the mind, carrying a poor prognosis. It is certain that we know more about the sequelæ of injuries than we do about their causes.

It is possible that these more clearly recognizable traumatic syndromes are partly obscured, because injury may in fact bring into being a whole range of psychological states, varying from the obvious psychotic results of direct physical damage to the brain and the acute traumatic deliria, right through to clear examples of schizophrenia and manic-depressive psychosis. Because these obvious endogenous psychoses occur in association with the injury, it is thought by the patient, by his relatives, and by the officials and tribunals, that the injury is wholly responsible for the psychosis it precedes. This is an extreme example, but will serve to remind you of events which, at some time or other, you will all have encountered.

Sir David Henderson, in his classical textbook, has drawn some distinction between the traumatic neurosis following physical trauma and the traumatic psychoneurosis due to the various psychological effects activated by the physical damage. Notwithstanding such distinctions, the extent or nature of the surgical injury is never the factor which alone determines the subsequent neurosis or neurotic disability. The important factor is the psychological predisposition to neurosis. In this context, the injury may be regarded as the precipitating event; and the categorical classification of the neurosis is of trivial importance compared with the clinical plan which should be put into action every time, whatever the specific psychological diagnosis.

We may, if we wish, preserve the term compensation neurosis, for in the space of two short words it perhaps conveys a good deal of clinical information to a fellow medical practitioner. But the symptoms and signs which make it up, and the prognosis it bears, are the same as in any neurosis. If there is anxiety or simple depression, the prognosis will be good; but if gross obsessional or hysterical features dominate the picture the prognosis will be relatively bad. I remind you of the reality of these several points by telling the following typical story.

A joiner, aged 45 years, fell down into a cellar. He was unconscious for a while, with a post-traumatic amnesia of some seven minutes; but all that was found on examination were several bruises and a fractured left clavicle. The man had a steady work record, and for that matter a steady life record. As his bone healed he was somewhat handicapped by limitation of movement in his shoulder. This, he soon said, affected his chest and head. It was but a small step for him to complain of headache, constant headache and giddiness. The lack of power in his arm then became increasingly worse at subsequent examinations, and even spread to his other arm, while surgical and neurological examination showed fewer and fewer signs to account for it. We did not leave the case at this juncture, but instituted a full but very simple psychological investigation.

A very brief summary of the further inquiry is that on the day after the accident he was to have started on a new assignment. To see what this amounted to, he had opened the door to the cellar where he was to work and so fell down. No one had told him of the drop, and he still resented this many months after the accident. At an early medical examination he had been told that there was nothing that medical treatment could do for him, and that he should find himself a lighter job. He rejoined that he was a trade union man, and that so long as his compensation money was coming through he would wait until he was fit to resume his own job. Another job was out of the question. He had a grievance against the hospital because he was not seen by a particular doctor. He also had a further grievance against his union colleagues because they worked extra overtime when they had previously conspired together not to do so. Further, he spontaneously mentioned that he had not been fit for service in the last war on account of effort syndrome, and he had continued to suffer from this long after the war had ended. Of his previous history and personality it is worth noting that he had never had much money to spend on himself, largely through the mismanagement of his wife (who gossiped a lot), and also because all his three daughters had gained scholarships to a school where it was increasingly expensive to maintain them and to pay for their extra necessities. The feeling that he deserved much rest, special consideration and monetary compensation greatly influenced his recovery, and completely set the stage for his behaviour in the subsequent course of his illness.

With this example I may now state the main theme of my argument—the multiple ætiology of the compensation syndrome and the consequent multiple approach to its treatment. Through the surgeon's eyes the surgical injury rightly occupies the centre of the list. But in every case (and not just in a few specially selected for their psychiatric teaching value), surgical, psychological and sociological, intellectual and economic, cultural and industrial factors all play their part in determining the clinical course after injury, even though in many cases the part of each factor may be a minute one.

It follows that our clinical plan must be to investigate all these factors. This involves a very changed approach. But I think you will concede that by changing our approach to an already recognized disease we are often able to bring about as much advance as if we discover an entirely new disease. For so long as we regard tuberculosis simply as a disease of its bacillus, so we arrange our treatment exclusively round the stamping out of that bacillus; but if we regard clinical tuberculosis as a disease of the person in some more total sociological way, so shall we orientate our treatment to those ends.

Personal Factors.

Of course extremely large injuries make the prognosis worse than extremely small injuries. Extremes, like statistics, can be used to prove or disprove any hypothesis. But for those of us who have been brought up to assume that the lesion has a lot to do with recovery, it may be a bitter pill to swallow to recognize that in the general run of injuries it has practically nothing to do with it. Some recent figures from Richardson and Weir (1957), though taken in a different framework of inquiry, will serve to illustrate this very clearly. Patients were divided into

those with severe, those with moderate, and those with slight disability. After six months, of those with severe disability 50% were back at work; of those with moderate disability 47% were back at work; and of those with slight disability 48% were back at work. That is to say, the figure is roughly 50% in all three groups. A number of factors overlap with this, but at the least the figures show that the severity of the disability is not the determining factor.

Let us then consider one or two factors which do determine the subsequent prognosis. Age is one of these. If you follow up the patients occupationally, you will find after some months that of men under the age of 40 years, 70% will be back at work. Of men over 50 years, only some 30% will be back; while between these two middle ages approximately 50% will be back in employment. The man's age is thus more important than his specific disability. So is his skill.

If we again divide up our workers by their occupation, calling them skilled, semi-skilled and unskilled, we find a comparable state of affairs. We find that over 60% of the skilled and semi-skilled men return to work in approximately the same time as do 40% of the unskilled. Of course, the possession of a skill no doubt reflects a number of personal qualities which have led that man to acquire that skill, and it suggests in general that he is likely to be better motivated and intellectually and emotionally better equipped than those less skilled. However, the fact that more general factors lie behind the possession of a skill in no way detracts from the value of using this as one indicator of these qualities in seeking such practical correlations as are here under review.

This leads to my next point; and to this Richardson has also paid attention. In the studies in these fields undertaken by the experimental psychologists, what has emerged as the most important prognostic indicator is what they refer to as attitude. The patient's attitude to work is rated at the time of injury on a carefully constructed scale. In this study there were 50 patients whose attitudes were good, and 80% of these, when they were followed up six months later, were found to be back at work. In striking contrast, of all those patients whose attitude was poor, only 20% were back. Of course, in this context, what the lay psychologist is appraising when he uses his rating scales is something probably not so very different from what we should include as one of the main neurotic parameters in our discussion of compensation neurosis as a clinical syndrome. Such studies will often take the form of expressing in clear experimental language some of the findings which frequently occur clinically. Whatever the correlations are, a ratio of 80% to 20%, four to one, is a clear indication of the importance of the patient's "set" toward his work.

Over and above this we must look for any ordinary specific psychoneurotic tendency, specific to this patient in this illness. One man who had sustained a severe head injury in the course of attempting suicide was off work for many months because of the continuing depressive illness which had led to the suicidal attempt. A boy with only a momentary amnesia after a trivial injury persisted for weeks with reasons why he should remain away from work, and a full history revealed that he had been terrified at the time when he was squeezed between a bus and a lorry. It amounts to this: when we are assessing the results of an injury we must take into consideration the whole gamut of factors referable to that patient in his social context. That sounds simple enough. In one or two isolated clinics (which are famous for it) this is done, but in most nothing more than lip service is paid to the importance of this approach.

The first main point of my thesis, then, is that for effective therapy we must really take them into account, not just for purposes of fanciful theory, but for the satisfaction of hard clinical facts. And I have given you figures to prove the point.

My second, and less important point is that over and above this there are usually particular features which affect treatment and prognosis, such as the specific psychoneurotic depression or phobia just cited; or, for example, the duration of post-traumatic amnesia in cases of concussion. These and many more points belong, as it were, to my second level of clinical inquiry; but these important oddments are in practice, in good hospitals, rarely overlooked.

Social and Political Aspects.

Now we come to the third level of inquiry: the one which has always lurked so apologetically in the background, but the one which is so important. From our current social context there is one feature, once unknown, which today is disturbingly common and which is every day increasing. This is the idea that a man must be fit or not fit; the idea that he must do his usual routine union job, or not do it; the idea that he must have a doctor's certificate saying so, one way or the other, yea or nay. And all of this apparently to protect his social rights, but actually (you and I may think) to fit him neatly into the administrative requirements of our new social benefit schemes and into the working rules of our politically determined working conditions. His employer and his fellow work mates (and, of course, the patient who is one of them) do not want him to return until he is fit to do his usual trade union job. They will rarely accept any of the possible intermediate compromises and have him back on work sympathetic and suitable to his partial incapacity. They will certainly not accept the idea that to have the patient back at work may make him well; and they do not listen to the notion that not returning to work may prolong the very maladjustment which is keeping him away. It can fairly be said that they all, including the patient, contribute directly to the hindering of his recovery and his return to full employment. It is a laughable but pathetic vicious circle. In many cases the contribution of this kind may be very small; but in others it is very considerable, and keeps the man away from work when his injury has long been forgotten.

Why, some of you may ask, do I bring up these uncomfortable considerations? The doctor should not go meddling into politics. The answer is that really he is not. He is but laboriously unravelling essential clinical factors which are of direct ætiological significance in the sequelæ and clinical management of industrial injuries today. To cure his patient he must know him. It is as much his duty today to examine (or, if you must have it, to interfere in) matters affecting the psychological health of his industrial patients as it was for John Snow in 1800 to interfere in the commercial supply of water to the City of London when he had amassed sufficient evidence to incriminate it in the fatal spread of cholera in that city; or of Legge and Goadby in 1900 to incriminate the lead industry when they had found its hazards fatal to its workers.

Surely, too, such considerations convince you that neurosis after injury cannot be equated directly with disablement after injury. The presence of neurotic symptoms and signs, or even of a full-blown neurosis, does not mean that the man will not work. Psychological complications, like the injury itself, are a very different matter from actual disablement, and are to be distinguished clearly from the subsequent industrial or social maladjustment, or other failure of personal resettlement, and the failure to resume work due to these social and economic barriers.

As part of our increased social attention to the general medical population, we delve into more and more psychosocial and psychosomatic factors. Surely as a particular example of this we should pay more attention to the same things in our workers? I should have thought that such attention to the workers was a primary consideration in the making of a welfare state, provided that other sections of the population do not thereby suffer. But the welfare-state-makers do not seem to think so. If, therefore, it is necessary for some of us—as physicians—to examine matters which are in the first instance non-medical, or political, we must still do so.

Summary.

The great economic as well as the clinical importance of neurosis in industry is discussed; and the influence of

compensation and other current industrial customs and non-medical factors is considered in relation to the events after industrial injuries.

The multiple ætiologies of the compensation syndromes are discussed with reference to a number of desirable changes in attitude.

Acknowledgements.

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SHIGELLOSIS IN VICTORIA.

By G. N. COOPER,

Public Health Laboratory, University of Melbourne, Melbourne.

WHILE acute bacillary dysentery is rarely encountered in Australia today, organisms belonging to the genus *Shigella* are frequently isolated during the routine diagnosis of enteric infections; these organisms are probably all capable, under certain circumstances, of producing the classical symptoms usually ascribed to *Shigella dysenteriæ*. Although many reports of the bacteriological, epidemiological and clinical implications of enteric infections caused by *Salmonellæ* in this country are available in the literature, there is a surprising dearth of information on the incidence of shigellosis. It is almost certain that these infections are as common to Australia as in many other countries, and they can on occasion present epidemiological problems of similar magnitude to those encountered with the *Salmonellæ*. In view of this, it seemed worthwhile to review briefly the available literature on *Shigella* infections in this country and to record the findings of the Public Health Laboratory, University of Melbourne, over the past six years.

It is perhaps unfortunate that for many years organisms belonging to the genus *Shigella* have been recorded under a variety of specific and generic names, and in many early papers it is not easy to determine the exact type of organism being discussed. However, recently a rational system of nomenclature has been established (*Int. Bull. Bact. Nom. and Tax.*, 1954, 4:1), and it is proposed to adhere to this system throughout the ensuing discussion. Where necessary, alterations will be made in the specific names used by previous authors.

In 1911 Forsyth recorded the occurrence of *Sh. flexneri* (type unknown) and of *Sh. dysenteriæ* type 1, in a number of children suffering from bacillary dysentery in Melbourne. This report contains the only reference to the occurrence of "Shiga's bacillus" (*Sh. dysenteriæ* type 1) in Australia. Paterson and Williams (1921) first reported the isolation of *Sh. sonnei* in Melbourne together with *Sh. flexneri* (probably type 2a); infections caused by the latter organism, however, were far more common than those in which *Sh. sonnei* was isolated. This was also observed by Beare in Adelaide two years later (1923). The prevalence of infections caused by *Sh. flexneri* type 2a apparently continued for a considerable period. Nelson (1932), while investigating a series of 122 cases of diarrhœa and dysentery among Melbourne children, recorded *Sh. flexneri* (probably type 2a) in 58% and *Sh. sonnei* in 3·3%, and in 9% of the cases an organism which was probably related to *Sh. boydii*. Harper (1932) at the same time recorded the isolation of 69 *Shigella* strains from a series of 206 cases of epidemic diarrhœa in Sydney, but no indication of the species distribution of these strains was given. In 1950 Mushin noted the isolation of 15 strains of *Sh. sonnei* from patients in the Royal Children's Hospital, Melbourne; 11 of these cases apparently arose as a result of cross-infection within the hospital wards. A comprehensive survey of *Salmonella* and *Shigella* infections in children was published by Williams (1951); in contrast to previous reports, he found *Sh. sonnei* infections to be far more common than those caused by *Sh. flexneri*, although the numbers may have been weighted by a series of cross-infections of *Sh. sonnei* occurring in hospital wards. However, this author emphasizes "the reappearance of *Sh. sonnei* in Sydney after many years of quiescence".

This present report is intended to survey the incidence of *Shigella* infections in Victoria over the past six years; the results recorded here do not include those obtained in surveys among mental institution patients, for it is considered that these present rather different epidemiological features from those observed in the general community. The problems of institutional *Shigella* infection will be discussed in a subsequent report.

Source of Strains.

The greater proportion of organisms recorded here were isolated from patients in the urban area; a number forwarded to the Laboratory for identification had been isolated from both in-patients and out-patients of the Royal Children's Hospital, Melbourne, while the remainder were isolated from fæcal samples received by the Public Health Laboratory. The remaining few included in this series were received for identification from a number of urban and country hospitals.

Isolation and Identification of Strains.

The strains were isolated and identified as follows.

Fæcal samples received in this laboratory were primarily cultured on desoxycholate citrate and "Difco" S.S. agar media, being incubated at 37°C for 24 hours. Initially tetrathionate broth was included (as an enrichment medium for *Salmonellæ*), but latterly, when *Shigella* infections were suspected, this was replaced by selenite F broth; this medium has been found to be satisfactory in encouraging the growth of *Shigella* in many instances. The enriched fæcal cultures were subsequently inoculated on "Difco" S.S. agar. Usually four to six non-lactose-fermenting colonies were selected for biochemical tests, being inoculated (by straight wire) into Kligler's iron agar, urea agar, mannitol peptone water and peptone water (for indole production). The identification of organisms producing reactions suggestive of *Shigella* species was subsequently confirmed by further sugar fermenting tests in lactose, sucrose and salicin peptone waters.

Final confirmation and identification were obtained by agglutination tests (both slide and tube methods) using boiled suspensions of organisms in mercuric iodide (1/1000) in physiological saline; specifically absorbed rabbit antisera, prepared in this laboratory and also supplied by the Standards Laboratory of the Public Health Laboratory Service (Colindale), were used for the antigenic characterization of the organisms. When necessary, strains were forwarded to Dr. K. Patricia Carpenter, of the Shigella Reference Laboratory, Colindale, for identification or confirmation of unusual types.

Results and Discussion.

Clinical Symptoms.

A variety of symptoms have been observed in cases recorded in this survey; these have included diarrhœa, the passage of blood and mucus in stools, vomiting, pyrexia, and on occasion convulsions. In several instances patients have presented with symptoms very similar to those encountered with *Salmonella* food poisoning; in two cases food was subsequently incriminated as the source of infection. Table I has been prepared to indicate the general range of symptoms, and records the various percentages of patients (infected with *Sh. flexneri* type 2a and *Sh. sonnei*) in four arbitrarily selected groups based on the clinical severity of the symptoms; these figures do not include patients about whom no satisfactory history was available.

Generally it would appear that the infections produced by *Sh. flexneri* type 2a are more severe than those due to *Sh. sonnei*; while only 6% of patients infected with the latter organism developed acute symptoms, no less than 43% of the patients suffering from *Sh. flexneri* type 2a infection were affected similarly (or more severely). Two children died as a result of these infections, but no deaths due to *Sh. sonnei* were recorded in this survey. The difference in severity of the infections is also borne out by the fact that in a total of 144 proven *Sh. flexneri* type 2a infections, 128 (88%) of the patients were admitted to hospital, whereas in 173 *Sh. sonnei* infections only 12 (6%) of the patients were admitted to hospital. However, as experience

has shown elsewhere, these results should not encourage the belief that *Sh. sonnei* is a relatively innocuous organism, for it will be shown in a later report that this organism is potentially capable of producing extremely severe symptoms; it is probably true that the severity of a *Shigella* infection depends not so much upon the particular species as upon the physical condition of the patient and other non-specific factors.

TABLE I.
Clinical Symptoms Produced by Shigella Infection.

Severity of Symptoms.	Percentage of Infections.	
	Sh. flexneri Type 2a.	*Sh. sonnei.*
Mild: Usually diarrhœa; occasionally blood in fæces, no pyrexia; recovery rapid..	12	43
Moderate: *Diarrhœa, blood and mucus in fæces,* occasional vomiting and some prostration, slight or no pyrexia; recovery rapid	45	51
Moderately severe: Diarrhœa, blood and mucus in fæces, vomiting, pyrexia; recovery slow ..	36	6
Severe: Diarrhœa, blood and mucus in fæces, vomiting, pyrexia, acute prostration, sometimes convulsions, occasional death; recovery slow	7	0

Incidence of Shigella Infections.

Table II records the isolations of *Shigella* species from the Victorian population over the six years from 1952 to 1957, inclusive. It is obvious that only two species, *Sh. flexneri* type 2a and *Sh. sonnei*, are endemic in this State, and have been responsible for the major proportion of cases of shigellosis during the period of this survey. The remaining types and subtypes of *Sh. flexneri* and the two types of *Sh. boydii* have been isolated on only rare occasions and apparently do not present any major epidemiological problems. Moreover, it is obvious from the figures presented

TABLE II.
Isolations of Shigella Species in Victoria, 1952 to 1957.

Species.	Number of Strains Isolated in the Year.						Total Isolated.
	1952	1953	1954	1955	1956	1957	
Sh. flexneri type 1a	—	—	—	—	1	1	2
Sh. flexneri type 1b	—	—	1	—	—	—	1
Sh. flexneri type 2a	58	37	23	14	11	1	144
Sh. flexneri type 3	1	—	1	—	—	—	2
Sh. flexneri type 4a	—	—	—	1	2	—	3
Sh. flexneri type 4b	—	—	—	—	—	—	—
Sh. sonnei ..	35	31	21	59	16	11	173
Sh. boydii type 10	—	—	—	—	—	1	1
Sh. boydii type 13	—	—	—	1	—	—	1
Total isolated	94	68	46	75	30	14	327

that the incidence of *Shigella* infections, based on the number of bacteriologically proven cases, has been declining over the period examined; the possible reasons for this will be discussed later. In contrast to the incidence of salmonellosis in this State (Cooper and Wilson, 1957), no significant seasonal fluctuation in the number of *Shigella* infections has been observed. While in the early part of the period surveyed there was a preponderance of *Sh. flexneri* type 2a infections in the community, latterly there has been a relative increase in the *Sh. sonnei* infections, and at present this organism appears to be the major cause of shigellosis. This parallels the reappearance of *Sh. sonnei* in Sydney recorded by Williams in 1951, although the change-over has apparently occurred later in Victoria than in New South Wales.

In any assessment of the importance of a particular type of infection in a community, the record of isolations of organisms provides relatively little information. Therefore, in Table III the number of incidents of *Shigella* infections

TABLE III.
Shigella Outbreaks in Victoria, 1952 to 1957.[1]

Year.	Number of Incidents.					
	Group 1. Sporadic Cases.		Group 2. Family Outbreaks.		Group 3. Institutional Outbreaks.	
	Sh. flexneri Type 2a.	*Sh. sonnei.*	*Sh. flexneri* Type 2a.	*Sh. sonnei.*	*Sh. flexneri* Type 2a.	*Sh. sonnei.*
1952 ..	30	14	5 (11)	1 (2)	4 (17)	2 (19)
1953 ..	30	9	1 (3)	3 (6)	1 (4)	1 (16)
1954 ..	19	8	1 (4)	1 (6)	0	2 (7)
1955 ..	12	17	0	7 (18)	1 (2)	3 (23)
1956 ..	9	8	1 (2)	3 (8)	0	0
1957 ..	1	7	0	0	0	1 (5)
Total	101	63	8 (20)	15 (40)	6 (23)	9 (70)

[1] Figures in parentheses indicate number of patients involved in outbreaks.

(due to *Sh. sonnei* and *Sh. flexneri* type 2a) are recorded; these have been arbitrarily classified into three apparently different epidemiological groups, as follows: group 1, sporadic cases, between which no conclusive connexion could be found; group 2, family outbreaks involving two or more persons who were living in the same household, or between whom close contact had been known to occur; group 3, institutional outbreaks involving two or more persons living under close communal conditions.

Besides the obvious children's institutions, hospitals, etc., which are classed in group 3, certain migrant hostels, holiday camps and housing settlements have also been included here; in such places community life occurs, and in some instances under conditions bordering on the unsavoury.

It is evident from this table that *Sh. flexneri* type 2a infections occur more sporadically than do those caused by *Sh. sonnei*; on the other hand, the outbreaks in families and institutions are more often caused by infection with the latter organism. While only one serious outbreak of *Sh. flexneri* type 2a infection (involving more than three or four persons) has occurred over the past six years, a number of outbreaks of *Sh. sonnei* infection (on several occasions involving 15 to 20 patients) have been observed in establishments where a close community life is led.

In most countries at the present time shigellosis is generally held to be a disease of the young. The figures recorded in Table IV indicate such to be the case in this

TABLE IV.
Age Incidence of Shigella Infections.

Age Group. (Years.)	Number of Patients Infected.	
	Sh. flexneri Type 2a.	*Sh. sonnei.*
Under 2	66 (46%)	53 (30·5%)
2 to 6	52 (36%)	46 (26·5%)
7 to 10	11 (7%)	22 (12·5%)
11 to 14	2 (1·5%)	13 (7·5%)
Over 14	13 (9·5%)	39 (23·0%)

State. The bulk of patients from whom *Sh. flexneri* type 2a was isolated were young children, while the infection was observed relatively infrequently in older children and adults. However, there is a far more equal distribution of infection among the various age groups with *Sh. sonnei*; almost as many adult infections were recorded during this

survey as those in infants under the age of two years. It is conceivable that this difference in age incidence between the two endemic organisms may explain the apparent differences in severity of infection recorded in Table I. As a general rule, the younger the child the more severe were the symptoms, and thus the greater number of *Sh. sonnei* infections in adults and older children, in whom the infection was generally mild, would tend to weight the figures recorded for *Sh. sonnei* towards the less severe type of infection.

Epidemiology of the Shigella Infections.

The problems of these infections in this State are by no means as great as those presented by the *Salmonellæ*; the total number of *Shigella* strains isolated over the past six years have been approximately one-third the number of *Salmonella* strains isolated from human patients over the same period (Cooper and Wilson, 1957). Nevertheless, shigellosis may still be considered one of the more common infectious diseases recorded here, and as such it presents its problems to public health bacteriologists and medical officers. The fact that since 1955 there has been a marked decline in incidence of these infections does not necessarily indicate that successful control has been achieved.

With the risk of stating the obvious, *Salmonella* and *Shigella* infections cannot be considered epidemiologically similar; this is due basically to the fact that whereas a large inoculum of *Salmonella* organisms (excluding *Salm. typhi*) is required to establish an enteric infection (Newell, Hobbs and Wallace, 1955), relatively few *Shigella* organisms are required to produce clinical symptoms (this is well evident from the relative frequency of infections among laboratory workers handling these organisms). It would appear that the pathway of a *Shigella* infection does not require an intermediate link wherein multiplication of organisms can occur, as do the *Salmonellæ* (Cooper and Wilson, 1957). Moreover, as there are no known reservoirs of *Shigellæ* other than humans and perhaps monkeys, many epidemiological problems encountered with the *Salmonellæ*, particularly those involving animal sources of infection, do not arise.

It is well established that *Shigella* infection generally results from direct human-to-human transfer (which would easily allow the exchange of the few organisms necessary to initiate infection), or indirectly via communally used articles which are subject to human fæcal contamination (e.g. water closets, bedpans, towels, wash basins etc.). Although the transmission of *Shigella* by flies and other infected vermin has been well established in army camps, etc., over a period of many years, it is considered that such vectors are of little importance in the transmission of these infections in civilian communities; this view is supported by Rubbo (1952). Hardy and Watt (1947), after a comprehensive survey of shigellosis in America, discount almost entirely sources such as flies, vermin, milk, water and other foods. As was mentioned previously, on only two occasions have foods been incriminated as a source of infection in this community; the presence of *Sh. flexneri* type 4a in sausages was established once, although its origin there remains unexplained. In the other instance *Sh. sonnei* was isolated from mussels obtained from the lower reaches of the river Yarra, where gross fæcal contamination (particularly from overseas ships) might be expected. Shell-fish regularly are contaminated by acting as "bacterial filters" in polluted areas, and the infection that resulted from consuming these mussels was not unexpected.

If the most common means of transfer of *Shigella* infections is by close personal contact, then it can reasonably be expected that the bulk of infections will occur among persons living under conditions favouring this; institutions, migrant hostels, holiday camps, perhaps also hospitals, with communal eating, bathing and toilet facilities, as well as individual homes are obvious places in which this may be expected. The figures presented above support this belief, as it has been shown, for instance, that the majority of *Sh. sonnei* infections have occurred as outbreaks involving two or more people at one time living in close contact. While *Sh. flexneri* type 2a infections do not support this view to the same extent, many of the cases recorded as "sporadic" have occurred in children living in housing settlements and migrant hostels.

It is very likely that indirect transfer of organisms, via communal toilet and bathing facilities which are subject to gross fæcal contamination, is a major cause of the infections. Hutchinson (1956) has shown quite clearly that in institutions where known carriers of *Sh. sonnei* were housed, this organism could be isolated regularly from toilet seats, presumably being deposited there by splashing of semi-fluid stools during flushing of the toilet; moreover, she has shown that these organisms persist on such sites for considerable periods without any decrease in viability. The problem of transfer by such means increases in any community where the majority of people possess only a limited knowledge of personal hygiene and cleanliness. Similarly, it has been shown that communally used towels (particularly the roller variety) may serve as inanimate vectors of these organisms (Rubbo, 1948). It is obvious, therefore, that the most satisfactory means of reducing such infections, particularly in ambulant communities, is not only to provide clean and well designed toilet and washing facilities, but also to encourage greater personal hygiene and cleanliness in the residents of that community. This, of course, need not apply only to large communities, but should also be encouraged in individual households.

Infection among infants and other non-ambulant patients probably arises through indirect contact or directly from the nurse; probably the most satisfactory means of preventing cross-infection is the application of a strict barrier-nursing technique. This principle can rarely be achieved outside hospitals; in many children's homes and institutions, as well as individual homes, the people nursing the infants are untrained and therefore unable to use such techniques. In these instances simpler and more easily accomplished methods of reducing the chance of cross-infection would be desirable; such measures will be mentioned in a subsequent report.

It is probably true that a considerable number of *Shigella* infections pass unnoticed in the general population; the common attacks of diarrhœa, "gastric 'flu" and other such mild enteric symptoms in adults are rarely reported to the medical practitioners, and even more rarely are subject to bacteriological investigation. A proportion of these may be due to infection with *Shigella*, and probably many of the sporadic cases of proven shigellosis may be epidemiologically linked with such subclinical infections. Probably the bacteriological investigation of such mild enteric upsets might provide important epidemiological information on the spread of these infections in the community. Temporary asymptomatic carriers probably also exist in the community and may serve as a source of infection.

The apparent decrease in the number of *Shigella* infections that has occurred over the past three years encourages the hope that these infections may eventually be controlled in the general community. Probably one major factor responsible for this is the progressive elimination of housing settlements and "slum areas" in the inner suburbs of Melbourne; such cleared areas at one time provided the bulk of the cases recorded. Hardy and Watt (1947) stated that "the main factors in *Shigella* outbreaks are poverty, overcrowding and a general personal lack of cleanliness". It is believed that the reduced incidence in this State indicates that these factors are gradually being overcome.

Summary.

1. The incidence of *Shigella* infection in Victoria over the past six years is recorded. *Sh. sonnei* and *Sh. flexneri* type 2a are both endemic in this community and have been responsible for the majority of these infections.

2. The severity of symptoms has varied considerably, but *Sh. flexneri* type 2a infections have apparently been more severe than those caused by *Sh. sonnei*. However, this may be related to the fact that infections due to the former organism were more commonly found in children and infants, whereas *Sh. sonnei* occurred very often in adults and older children.

3. Certain epidemiological features of shigellosis have been considered, and general measures for its prevention have been indicated.

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TROPICAL PULMONARY EOSINOPHILIA.

By JEKABS KARIKS,

Department of Public Health, Goroka, New Guinea.

THE purpose of this paper is to draw attention to a very interesting clinical condition in the Australasian region, tropical pulmonary eosinophilia, which is met probably frequently enough, but rarely diagnosed as such. The cause is simply lack of awareness of the presence of this disease.

The following are briefly the clinical symptoms and signs of tropical pulmonary eosinophilia (T.P.E.). Cough is the presenting symptom, and is always present, frequently with spasmodic attacks resembling bronchial asthma, hence frequently diagnosed as such. The cough may be acute or chronic, and is most severe during the night and in the early morning between 2 a.m. and 4 a.m. Cough may persist also during the day, but is usually of a less severe character. There is a scanty production of sputum. Other symptoms associated with this condition such as lassitude, fever, blood-streaked sputum, pain in the chest, dyspnœa, orthopnœa, glandular enlargements, enlarged spleen and liver, insomnia, sweating during the paroxysms, anorexia and loss of weight may or may not be present. When present, they have some diagnostic significance. Their absence does not exclude the diagnosis of T.P.E.

The ætiology of this disease is still regarded as obscure.

Physical findings are as follows. There are harsh breath sounds over both lungs, over one lung field only, or over the bases only, scattered rhonchi and crepitations. An X-ray examination of the lungs in typical cases reveals mottled shadows resembling closely those of miliary tuberculosis, for which they are frequently mistaken. In other cases an X-ray examination will show only markedly increased coarse striation over the lung fields. Hilar shadows may be enlarged. Sometimes some haziness over one or the other lung field may be the only sign. The total leucocyte count is always increased up to 30,000 or over. Danaraj (1947) has recorded a count of 100,000. The eosinophil count may go as high as 80% to 90%. The erythrocyte sedimentation rate is usually increased.

Treatment is by arsenical compounds such as "Acetylarsan", "Mapharsen" and neoarsphenamine. In very recent years diethylcarbamazine ("Hetrazan") has also proved to be a specific drug for use in the treatment of T.P.E.

To the previously described cases in the medical literature I should like to add five more. These are from my own observations made in New Guinea, New Britain and Bougainville.

Reports of Cases.

CASE I.—A male native about 40 years old, from the Rabaul area, New Britain district, was admitted to Rabaul Native Hospital in May, 1954. He complained of cough, pain in the chest, dyspnœa on exertion, and progressive loss of weight due to anorexia. All these symptoms had prevailed over the last four months prior to his admission to hospital. He complained also about insomnia due to the troublesome cough during the night. On examination the patient was emaciated and appeared to be in distress. Orthopnœa was present. He had very harsh breath sounds with multiple medium sized rales and rhonchi over both lung fields. His abdomen was soft, and there was no tenderness. The spleen was enlarged, the liver not palpable. No abnormalities were detected in all other systems, and there was no glandular enlargement. An X-ray film of the lungs taken on the same day revealed mottled shadows over both lung fields resembling those of miliary tuberculosis. He was then diagnosed as suffering from miliary tuberculosis, and the appropriate treatment with streptomycin, PAS and INH was initiated. Repeated examinations of sputum did not reveal any acid-fast bacilli. Fever, which was present on the first day of his stay in hospital, cleared and the patient was afebrile afterwards. His cough persisted. As a routine measure blood samples were examined and revealed a leucocyte count of 18,000, with 83% eosinophils. Further blood samples were examined a few days later, and this time *Wuchereria bancrofti* was present. As repeated sputum examinations yielded no acid-fast bacilli, the original diagnosis of miliary tuberculosis was questioned. On account of the finding of *W. bancrofti* in his peripheral blood, the patient received "Hetrazan" treatment. A few days later he felt much better. Cough and dyspnœa disappeared gradually. On consecutive X-ray films taken about three weeks later, marked improvement was recorded in comparison with the first film. The lung fields had become much clearer. At the same time the leucocyte count decreased to 12,000, with only 35% eosinophils. After about six weeks in hospital the patient was virtually free from his previous symptoms and left the hospital of his own accord. The initial diagnosis was changed to filariasis, and only later, retrospectively, to T.P.E. No other drugs besides "Hetrazan" were used for the treatment. The initial tuberculosis routine treatment was discontinued in the second week of his stay in hospital.

CASE II.—An adult male native about 30 years old, from the Rabaul area, New Britain district, was admitted to Rabaul Native Hospital at the end of May, 1954. He complained of persistent cough for a few months, during the night, fever, and swellings in the groins. On examination, wheezing breath sounds could be heard over both lungs. The spleen and liver were not palpable. There were tender lymph glands in both inguinal regions. The total leucocyte count was 19,800, with 45% eosinophils. A blood slide made at about midnight revealed *W. bancrofti*. An X-ray picture of the lungs revealed much increased fibrous striation over both lung fields. Treatment with "Hetrazan" was commenced. After two weeks' treatment all symptoms (e.g., cough, fever) had disappeared. Lymph glands were reduced in size, and there was no tenderness. No microfilariæ were present in the peripheral blood.

CASE III.—A male native about 20 years old, from the Rabaul area, New Britain district, was admitted to Rabaul Native Hospital towards the middle of July, 1954. He complained of cough, little expectoration, and he sometimes felt feverish. On examination, no abnormalities were detected in any systems, except for scattered rhonchi over the right apex. X-ray picture of the lungs revealed an enlarged right hilus with increased striation towards the right upper field. The total leucocyte count was 15,000, with 20% eosinophils. No microfilariæ were seen on repeated examinations. Treatment with "Hetrazan" was commenced. After about two weeks' treatment he was practically free from his previous complaints, and left the hospital of his own accord.

CASE IV.—A male native patient about 25 years old, from the Kieta area, Bougainville district, was admitted to Kieta Native Hospital in August, 1956. He complained of a troublesome cough during the night, a little expectoration and fever at times. On examination, wheezing breath sounds could be heard over both lungs, with scattered crepitations over both bases. He had an enlarged spleen. No acid-fast bacilli were present in the sputum. No X-ray picture was taken as facilities were not available. The total leucocyte count was 17,400, with 43% eosinophils. Blood slides were repeatedly taken during the night, and in one of them *W. bancrofti* was present. On "Hetrazan" treatment the patient's clinical condition improved satisfactorily. After about three

weeks' treatment he had just a slight cough and a few scattered rhonchi over the lungs. The total leucocyte count in the fourth week was 9000, with 6% eosinophils.

CASE V.—A European female patient, 35 years old, residing on Bougainville Island, complained of severe cough, lassitude, fever, pain in the chest, and insomnia due to the persistent cough during the night. These complaints had lasted for the past two weeks. On examination, very harsh breath sounds with multiple rales over both bases could be heard. No X-ray picture was taken. Stool and urine examinations showed no abnormalities. The total leucocyte count was 12,600, with 20% eosinophils. Sputum was scanty, with a few rusty streaks. Numerous eosinophils were present. For the first few days penicillin was given intramuscularly, and "Ticarda" cough mixture. The initial fever subsided. Due to some local circumstances no night blood was examined. On account of increased eosinophilia with persistent cough, "Hetrazan" treatment was given. From the second day on "Hetrazan" treatment the condition improved markedly, and after two weeks' treatment only a slight cough was present, and this ceased completely after another two weeks of continued treatment with "Hetrazan" in reduced dosage.

Discussion.

The diagnosis of the first case described was not clear. The second patient was treated for filariasis, and the benefit of this treatment upon the persistent cough was noted. Only the last three patients were treated purposefully after the appropriate literature had been studied, and the diagnosis of T.P.E. was made at first hand. Arsenicals were not used for the treatment as purely by chance "Hetrazan" was found to be satisfactory, and I wanted to explore further its use for treating T.P.E. Later studies of the literature revealed that diethylcarbamazine, of which "Hetrazan" is the commercial name, has been found effective for the treatment of T.P.E. elsewhere (Jarniou *et alii*, 1957, Danaraj, 1947).

Confusion persists in the description of T.P.E., as there is no agreement as to the common terminology of this particular clinical condition. As long ago as 1950 Ball pleaded that the name tropical pulmonary eosinophilia be universally accepted. However, this and other conditions associated with eosinophilia in the tropics are still described under a variety of names such as tropical eosinophilia, eosinic pneumopathia, pulmonary eosinophilia etc. This leads to more confusion and uncertainty as to the condition described. In the tropics there are many causes which lead to eosinophilia with different clinical manifestations, e.g., all kinds of helminth infestation, coccidioidomycosis etc. A few factors put T.P.E. on its own: the lungs are always affected, causing very particular symptoms, the condition is not transitory and it needs very specific treatment. Increased leucocytosis, with a high percentage of eosinophils, is always present. The specific and effective treatment will confirm the diagnosis, if in doubt. Therefore the term tropical pulmonary eosinophilia is self-explanatory and best fits this condition.

Controversy as to its ætiology has persisted, and the question may not be answered for a while yet. Such agents as virus (Diaz *et alii*, 1951; Reisel and Groen, 1951), spirochætes (Treu), "visceral larva migrans" (Gault and Webb, 1957), Tyroglyphid mites (Koumans, 1957), *Fasciola hepatica* (De la Riva, 1949) and microfilariæ (Reisel and Groen, 1951; Malhorta, 1950; Kouwenaar, 1948) have been named as the causes. It would lead too far, however, to discuss all the advanced arguments in favour of each of these agents. The five cases described leave very little doubt that *W. bancrofti* was the primary causative factor in this series. The following points in favour of microfilariæ as being the causative factor in T.P.E. should be considered:

1. T.P.E. is a disease confined to the tropics.

2. T.P.E. has been described from the following places: (*a*) India: cases mainly described from the southern parts of India and Ceylon. No cases have been described yet from Pakistan and other northern provinces of India. (*b*) Far East: cases have been described as occurring in Burma, Siam, Java, South Coast of China mainland, Korea and Japan. (*c*) Australasian region: Queensland, Samoa and Fiji. (*d*) Africa: Egypt, Tanganyika, Uganda, Congo and Nigeria. No cases so far have been described from South Africa. (*e*) America: Rio de Janeiro, British Guiana, Curacao, Porto Rico and Cuba. (*f*) Europe: a case has been described from Italy occurring amongst the Polish troops during the war. A single look upon the world map showing the distribution of filariasis reveals that it covers exactly the areas from which T.P.E. has been described. From the northern parts of India and also from the South African region no case has been described. These areas are also free from filariasis. The case described by Broun *et alii* from Italy will be understood more clearly if the troop movements through the North African region, prior to their landing in Italy, are taken into consideration.

3. Hawking and Thurston have given evidence that the microfilariæ are concentrated mainly in the fine capillaries and blood vessels of the lungs when not present in the peripheral circulation. McFadzean and Hawking (1956), in reporting upon the periodicity of microfilariæ, also make the following statement: ". . . and there is an active phase in which the microfilariæ are concentrated in the lungs. The purpose of this active phase is less clear, but we assume that the lungs are the optimum site in the body for the survival of microfilariæ."

4. Microfilariæ do assemble eosinophils in great masses around them. Maier (1949) first described this phenomenon with *Loa loa* and *Acanthocheilonema perstans*. Schmidt and Dessanayake (1950) have since described a similar phenomenon with *W. bancrofti* in Ceylon. This phenomenon may help to explain the typical mottling shadows in the lungs, and explains also the great masses of eosinophils seen in the sputum in patients with T.P.E. Viswanathan (1947) has described the only post-mortem finding of T.P.E. in a man who died from arsenical encephalitis after treatment of T.P.E. with an arsenic compound. There were massive eosinophil concentrations around the bronchioles, though he did not find microfilariæ in these infiltrates. Similar concentrations of eosinophils have been described in the lymph glands of biopsy specimens with microfilariæ in their centre (Lie Kian and Bross, 1950; Hartz and Van der Sar, 1948). In none of these cases have microfilariæ been demonstrated in the peripheral blood.

5. Danaraj *et alii*, 1957, showed that patients suffering from T.P.E. gave a positive complement fixation reaction with extracts made from microfilariæ, although no microfilariæ had been found in the peripheral blood. Control patients, not suffering from T.P.E., did not give positive reactions.

6. Inorganic arsenical compounds are specific for the treatment of T.P.E. Arsenical compounds have been used also with good results for the treatment of filariasis (Otto *et alii*, Thooris, 1957). Diethylcarbamazine ("Hetrazan") is a specific for the treatment of filariasis. This same remedy has proved also very valuable in the treatment of T.P.E. (Jarniou and Moreau, 1957; Danaraj, 1947 and 1957; own observation).

7. In the past there have been described cases in which filariasis has also caused T.P.E. This applies especially to French and Dutch observations in Indochina and Indonesia.

8. Frequently described, but otherwise completely unexplained, is the enlargement of lymph glands in cases of T.P.E. In filariasis, lymph gland enlargement is a very frequent finding.

How does it come then that T.P.E. is described and observed so seldom whereas filariasis is encountered much more frequently in the countries mentioned earlier? The true explanation undoubtedly will need some more detailed investigations. I would like to make here only a few suggestions.

1. Only a minor fraction of all patients treated for acute or chronic bronchitis throughout the tropics reach the X-ray department or laboratory.

2. In most instances, as soon as signs of filariasis are seen and microfilariæ demonstrated in the peripheral blood, frequently also without it, the treatment for filariasis is given without any further investigation of the patients' condition or symptoms. This treatment probably also cures a few patients with T.P.E.

3. In the past, throughout the tropics, arsenical compounds have been used frequently and liberally for "blood purification". This practice too might have cured a few patients with T.P.E., besides other conditions, and hence also the widespread belief that neoarsphenamine is a good "blood tonic".

4. Some kind of allergy to microfilariæ and their products of metabolism is necessary to cause the symptoms of T.P.E. The asthmatic cough and the lung signs are very suggestive of this. As allergic reactions are seldom seen amongst the native peoples of tropical countries, this might explain why not every patient suffering from filariasis develops T.P.E.

5. In many cases of T.P.E. no microfilariæ have been found in the peripheral blood stream, but the same applies in many obvious cases of frank filariasis. Why this is so needs further investigation. The microfilarial complement fixation reaction might prove of great assistance in solving the question of the ætiology of T.P.E.

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THE PROBLEM OF APPREHENSION IN THE RH-NEGATIVE MOTHER.

By VERA I. KRIEGER, D.Sc., F.R.A.C.I.,
From the Department of Pathology of the Royal Women's Hospital, Melbourne.

IN 1941 Levine demonstrated that erythroblastosis of the new-born was due, in at least 90% of cases, to an incompatibility between the Rh-positive blood of the baby and the Rh-negative blood of its mother. Very few medical discoveries have been published so widely in the Press, and have caught the imagination of the lay mind to a comparable extent. The present almost universal practice of investigating the Rh status of the mother's blood early in pregnancy and the frequent testing for antibodies during the last trimester have contributed to an apprehension amongst Rh-negative mothers and their husbands, which is often quite out of proportion to the actual risks of such pregnancies. It has been stressed in many publications that only one child in about 200 might develop erythroblastosis, but there are only a few readily accessible figures at the disposal of the medical practitioner which he could use in order to alleviate such fears and apprehensions in his patients. We have therefore reviewed the outcome of pregnancies of Rh-negative mothers over a period of nine months at this hospital. The results are shown in Figure I.

Of 1635 Rh-negative mothers, 1533 had normal babies (94%), 102 (6%) had affected ones, and 16 (only 1·0% of the whole series) had either stillbirths or babies who died within a few days. Some of the 86 affected but surviving babies were only mildly affected, but many had to be treated with one or more exchange transfusions. In the group of 102 children who developed erythroblastosis, there was only one from a first pregnancy. It is necessary to stress the fact that whilst the first child of an Rh-negative mother and Rh-positive father usually escapes hæmolytic disease, transfusion of a young girl without regard to her Rh status may endanger her chance to have one unaffected child.

The 1533 unaffected babies included 672 (41% of the whole group) who could not be sensitized because they were Rh-negative, but also 861 (53%) who were Rh-positive. One quarter of these escaped hæmolytic disease because they were first babies, but 616 born in the second or later pregnancy were unaffected.

The blood of 11 women who were carrying Rh-negative babies contained antibodies. These were due to persistence of immunization from a previous pregnancy, in which the baby was Rh-positive, or to a preceding blood transfusion with Rh-positive incompatible blood.

The figures recorded indicate upper limits of the occurrence of erythroblastosis. In an average population they would be still lower, since a considerable number of Rh-negative women are admitted to our hospital when the outcome of previous pregnancies has been unsatisfactory. In addition, pregnancies in immunized mothers who have had a baby with hydrops fœtalis or a stillbirth in the preceding pregnancy are now often terminated at an earlier stage to try to prevent severe damage to the fœtus. It is therefore expected that our figures for severely affected or fatal cases will show a further decrease.

The results of this investigation can be summarized for easy reference as follows. Of 100 babies born to Rh-negative mothers, 41 will be Rh-negative and therefore unaffected; 59 will be Rh-positive, but of these 53 will be unaffected, six will show signs of erythroblastosis, but five will recover with adequate treatment, and only one will die.

In order to detect the few cases in which the baby will be affected and give these babies the greatest chance of survival as normal healthy children, it is necessary to determine the Rh status of all pregnant women and to check the blood of the Rh-negative mothers for antibodies

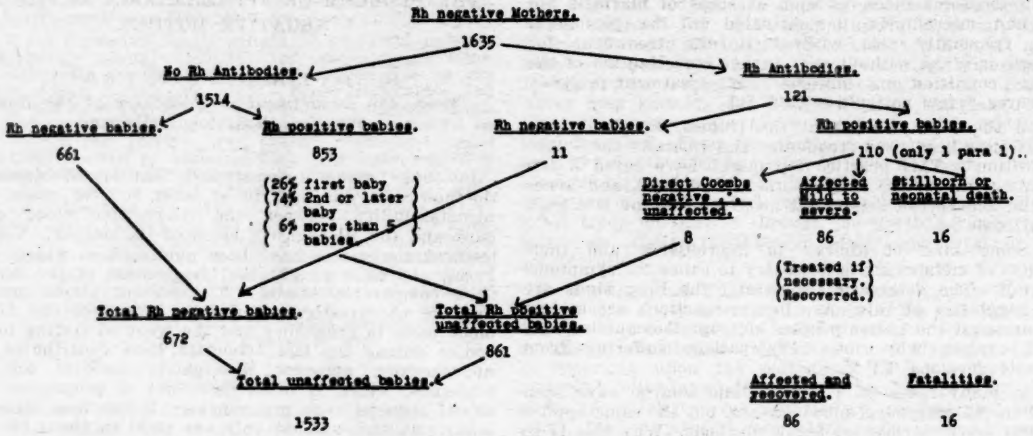

FIGURE I.

at intervals during the pregnancy. The performance of these tests with some explanation of the Rh story should not cause undue apprehension in the patients or their husbands if facts such as those presented are quoted to them.

Reviews.

Food for the People of Australia. By R. C. Hutchinson, B.Sc., D.Sc.; 1958. Sydney, London, Melbourne, Wellington: Angus and Robertson. 8¼" × 5¼", pp. 262, with illustrations. Price: 32s. 6d.

THERE are many ways of writing books about food. A new approach is given in this book. It is not a general text-book on food and nutrition, although all aspects of food requirements for normal people are dealt with adequately and in an interesting manner. The book is, as its title suggests, an able account of the food consumption pattern of the Australian people. There is an account of the food supply available and of the difficulties met in keeping up the supply to adequate levels from the beginning of settlement in Australia until the end of World War II. Government documents and reports of various committees have been used freely, and much interesting data have been made available. Certain aspects of the food consumption pattern in Australia are dealt with in detail, such as excess calorie consumption, excess consumption of sugar, the high intake of meat and the low consumption of calcium. The author gives a very full account of calcium distribution in foodstuffs and the reputed requirements of calcium by man where he lets himself be too much influenced by British and American figures for these requirements. Excellent and clear food consumption tables are given with data for planning adequate diets.

This is an excellent book on nutrition and very interestingly written. It can be thoroughly recommended for anyone, medical or not, who wishes to obtain a clear picture of nutritional matters in Australia. There are 32 half-page plates mainly on different aspects of the commercial preparation of food. These are in themselves interesting, but have little connexion with the text of the book.

Textbook of Virology for Students and Practitioners of Medicine. By A. J. Rhodes, M.D., F.R.C.P., F.R.S.C., and C. E. van Rooyen, M.D., D.Sc., M.R.C.P., F.R.C.P.; Third Edition; 1958. Baltimore: The Williams and Wilkins Company. Sydney: Angus and Robertson, Limited. 9" × 5¾", pp. 660, with 75 illustrations and 22 tables. Price: £5 10s.

THIS book aims to present the essential features of viral and rickettsial disease to students of medicine, bacteriology, virology and public health. The 1958 edition is certainly warranted because of the rapid advance now being made in this field. Even so, the enthusiastic reader would do well to consult recent literature, especially in the application of tissue culture to virus study and the investigation of agents such as E.C.H.O. virus, adenovirus and trachoma virus.

The subject matter—more clinical than scientific—is presented with simplicity and clarity, primarily for the student of medicine. The book does not aim to be an exhaustive review of the properties of viruses. References are few, and additional reading lists are appended to each chapter. The subject index is well arranged and comprehensive. The contents are divided into 12 sections—viz.: (i) the fundamental characteristics of virus infections of man and animals, (ii) skin diseases, (iii) exanthemata, (iv) respiratory diseases, (v) venereal diseases, (vi) eye diseases, (vii) arthropod-borne and tropical fevers, (viii) infectious hepatitis and serum jaundice, (ix) diseases caused by the Coxsackie viruses, (x) encephalomyocarditis and encephalomyelitis of animals, (xi) neurotropic virus diseases, (xii) rickettsial infections.

There are more than 80 excellent illustrations, which cover not only virus morphology, but technical procedures such as tissue culture techniques, mouse inoculations, chick-embryo techniques, serological tests, the use of the ultracentrifuge and electron microscope, clinical features and pathological lesions. These will be found exceedingly useful by those engaged in virus diagnostic work in the laboratory.

Poliomyelitis viruses are discussed in the "neurotropic virus disease" section, together with rabies, E.C.H.O. and encephalitis viruses. The Coxsackie viruses have a section to themselves. Thus, although the respiratory diseases are grouped in one section, there is no section on "enteric viruses" as such.

Books Received.

[The mention of a book in this column does not imply that no review will appear in a subsequent issue.]

"Advances in Internal Medicine", Volume 9, edited by William Dock, M.D., and I. Snapper, M.D.; 1958. Chicago: The Year Book Publishers, Incorporated. Melbourne: Ramsay's Medical Books. 8¾" × 5¾", pp. 400, with illustrations. Price: £4 13s. 6d.

Contains six articles on modern medicine.

"British Empire Cancer Campaign: Thirty-Fifth Annual Report Covering the Year 1957"; Supplement to Part 2. Report by Percy Stocks, C.M.G., M.D., F.R.C.P., on "Cancer Incidence in North Wales and Liverpool Region in Relation to Habits and Environment". 1958. London: British Empire Cancer Campaign. 9¾" × 7", pp. 170. Price not stated.

The titles are self-explanatory.

"Carcinoma of the Breast", by G. D. Haagensen, M.D.; 1958. Chicago, Illinois: Lakeside Press; R. R. Donnelley and Sons Company. 9¼" × 6", pp. 128, with 54 illustrations and nine tables. Price not stated.

The fifth of a series on the early recognition of cancer.

The Medical Journal of Australia

SATURDAY, DECEMBER 6, 1958.

SOLDIER-DOCTORS.

DURING this year the Royal Army Medical Corps has been celebrating its Diamond Jubilee. It was on June 23, 1898, that Queen Victoria signed the Royal Warrant which brought the Corps into being, and this Journal joins with many others in offering its congratulations not just on sixty years' survival, but on a record of ever-increasing distinction. A picture of the Corps and some of its outstanding figures has been drawn recently by a modest author signing himself R.E.B.,[1] and its history was traversed briefly by A. M. McIntosh[2] some years ago in this Journal; so we need not go over the same ground here. It is enough to mention that short work is made in these articles of the fallacious belief that a soldier cannot be a good doctor or a doctor an efficient soldier. Healing Apollo and destructive Mars would seem strange partners on the surface, but history shows that the partnership has been not only possible but fruitful.

Armies are as old as man, or at least, and ironically, as old as civilization. Medicine in some form is just as old. The true soldier-doctor is a relatively modern phenomenon, but armies have had their medical attendants for at least two thousand years. In the Roman armies they were non-combatant officers, and, according to Castiglioni, every legion and every warship in Hadrian's time had its own physician. The British and Continental armies and navies of more recent centuries have contained many distinguished figures, and none greater than Ambroise Paré (1510-1590), who was one of the pioneers of modern surgery, or James Lind, who first conquered scurvy. The list of notable doctors in recent times who found opportunity and fame through the army, navy or air force is long, ranging from Ronald Ross and W. B. Leishman to those whose work on malaria and other tropical diseases made a major contribution to victory in the Pacific area in the second World War. The great number of those who have been effective medical officers and soldiers, as distinct from doctors in uniform, is not so widely recognized as it should be, but these have become increasingly important as the organization of the medical services has grown in size and complexity. In this respect two interesting quotations appear in R.E.B.'s article. The first is from the report of the British Medical Association's Parliamentary Bills Committee of 1897: "Army medical officers must be something more than doctors, must not entirely cure diseases but prevent them, not merely professionally aid the wounded but take military charge. . . ." The second is from a leading article in *The Times* of August 10, 1951: "Army doctors belong to two professions, and unless they have mastered both they fail in their duty." It is beyond question that a great many, in both regular and citizen forces, have not failed in this duty.

An interesting symbolic light is cast on the doctor-soldier relationship in a study of armed forces medical insignia by Joseph Hirsh.[1] He has pictured and described a selected group of the insignia worn by the medical services of armies, navies and air forces throughout the world. The commonest symbol is, as might be expected, the serpent of Æsculapius, but the insignia vary from "such disparate forms as the Caduceus of Mercury, the Geneva or Red Cross, variations of the Maltese Cross, to singularly unmedical or unreligious symbols as stars on a background of cloth and the crossed rifles of the infantry". Popular insignia are the leaf and acorn derived from the Druid physician-priests of early Britain. More belligerent symbols, in addition to the crossed rifles already mentioned, are a sword and the Club of Hercules. Prominent among the insignia is that of the Royal Australian Army Medical Corps with the following description:

> It consists of a golden serpent entwined about a rod as the center-piece, surrounded by a circle bearing the legend: ROYAL AUSTRALIAN ARMY MEDICAL CORPS. This circle is in turn surrounded by a laurel wreath from which emanate an eight-pointed star of rays. Topping the insignia is the Royal Crown. At the base is the legend: PAULATIM.

The origin of this curious motto "*Paulatim*" is given by A. M. McIntosh[2] in a quotation from an address given to the United Service Institution in 1893 by W. D. C. Williams, who was then Principal Medical Officer in the Medical Staff Corps and was to be responsible for the organization of the Australian Army Medical Corps shortly after Federation:

> Regimental traditions are the sheet anchor of that national pride which encourages a soldier to look to his own regiment or service as second to none under arms—and when we consider the slow growth of the medical services, not only in the Colonies, but all over the world, sprung from seed sown on none too favourable ground, badly cared for in their early growth, occasionally pruned to such an extent as to cut them down altogether, may I suggest a motto which conveys in one word the life history and growth of the Medical Services and could most fittingly be adopted. I give you "Paulatim"—little by little.

It must be admitted that "*Paulatim*" has continued to be all too appropriate as a motto for the Australian Army Medical Corps (it became a Royal Corps in 1948), except during the periods of the two World Wars. Its progress has been a constant struggle. This is not for a moment to be regarded as a criticism of the small band of medical officers who have maintained their Corps in peace time; an appropriate motto for them might well have been "Much by Few". The fault, largely unwitting, has been first with those in government and service administration who have failed to appreciate the needs of the medical services of the armed forces and the attitude of civilian doctors when asked to entangle themselves in the machine of the services, and secondly with the members of our own profession who have been unable to see beyond the end of their clinical noses. The profession as a whole, and particularly through the British Medical Association, has always adopted a most sympathetic attitude to the medical services of the armed forces. In a "Commemorative Oration

[1] *Brit. M. J.*, 1958, 1: 1472 (June 21).
[2] M. J. AUSTRALIA, 1948, 1: 738 (June 12).

[1] *Mil. Med.*, 1958, 122: 256 (April).
[2] M. J. AUSTRALIA, 1948, 1: 491 (April 17).

on the Occasion of the Diamond Jubilee of the Royal Army Medical Corps", delivered in the Great Hall of B.M.A. House, Tavistock Square, London, on June 25 of this year, Sir Gordon Gordon-Taylor[1] laid emphasis on the important part played by the British Medical Association in the birth of the Royal Army Medical Corps. It appears that the Association was the midwife at this event, having in January, 1898, sent "a powerful deputation of representatives" to wait on the then Minister of State for War, as the direct result of which came the Royal Warrant to which we have already referred. The British Medical Association in Australia is no less sympathetic and has shown by its past and its current activity that it is ready to do what it can to ensure effective medical services in the armed forces and acceptable conditions for those who serve in them. It is hoped that negotiations now going on will greatly improve the prospects of serious medical work for young medical officers going into the services and will remove a major objection to recruitment of doctors. We may well hope then that our younger colleagues in their turn will respond to the opportunity, not just because it offers a good medical job or even because it is a reasonable service to their country, but because they seek new horizons.

Current Comment.

TROPICAL PULMONARY EOSINOPHILIA.

ELSEWHERE in this issue Jekabs Kariks, in describing a short series of cases of tropical pulmonary eosinophilia, puts forward an interesting argument that this obscure condition is in fact an unusual manifestation of filariasis, probably of the nature of an allergic reaction to the presence of microfilariæ in the lungs. This is a view which, as Kariks points out, has been gaining ground in recent years, though there are some serious difficulties against its acceptance. One of these is that though filariasis is an exceedingly common disease over large areas in the tropics, tropical pulmonary eosinophilia remains a rare condition. Kariks counters this with the suggestions that the condition may be very much commoner than is generally supposed, and that tropical races are little prone to allergic reactions. However, an annotation on the same subject in a recent number of *The Lancet*[2] reports an important new observation bearing on the question. It had already been suggested that tropical eosinophilia might be due to infection with filarial parasites of animal origin, which could not complete their development in man, as he was not a suitable host; hence the absence of microfilariæ in the peripheral blood of many patients with tropical pulmonary eosinophilia. It has now been reported that, in an investigation into the possible existence of animal sources of human filariasis, a human volunteer was inoculated with filarial parasites of animal origin, on one occasion from a monkey, on another occasion from a cat. On each occasion this hardy volunteer developed a cough and increasing eosinophilia, though no microfilariæ were found in his blood. In the second experiment the symptoms were cut short by a course of diethylcarbamazine.

Clearly the problem is not yet solved. In an area where filariasis is endemic, multiple infection with parasites of both human and animal origin is not unlikely. If tropical pulmonary eosinophilia is due to infection with parasites of animal origin, this would explain the presence of microfilariæ of *Wuchereria bancrofti* in the peripheral blood of some patients. On the other hand, tropical pulmonary eosinophilia may be a manifestation of a reaction which can be excited by filarial parasites of any kind, under special circumstances not yet elucidated. Whatever the ultimate explanation, it is difficult to visualize one which would account for the curious observation that, in Malaya, tropical eosinophilia is virtually confined to that section of the population which is of Indian descent.

[1] *J. Roy. Army M. Corps*, 1958, 104:116 (July).
[2] *Lancet*, 1958, 2:737 (October 4).

BLOOD GROUP ANTIBODIES AND RED CELL DESTRUCTION.

IN the last few years a considerable amount of work has been done on two aspects of the problem of incompatible transfusion reactions. The characteristics of different types of blood group antibodies *in vitro* have been studied in detail, and the renal complications of hæmolytic reactions have received a good deal of attention so that their pathological basis is fairly well known. However, another aspect of the problem which has received less attention is the manner in which red cells are destroyed in the body after contact with the appropriate antibody. This is poorly understood except when the antibody concerned has hæmolytic properties. We know that, in that case, the erythrocytes are hæmolysed in the circulation with the aid of complement. However, hæmolytic properties can be demonstrated *in vitro* only in a limited number of antibodies, of which immune antibodies of the A B O system and a few antibodies of the Lewis and P systems are outstanding examples. When other types of antibodies are involved, other mechanisms must be invoked to explain intravascular hæmolysis. At first sight, a study of how red cell agglutinates can be destroyed in the circulation might appear rewarding, for even if the antibody concerned is of the incomplete variety, the plasma constitutes the colloid medium necessary for agglutination, and therefore, in most cases, red cell agglutinates will be present in the circulation. However, even if the mode of destruction of agglutinates could be determined, the picture would still be incomplete, for hæmolytic transfusion reactions, sometimes with definite signs of intravascular hæmolysis, are reported from time to time, when the antibodies concerned are of a type that coats erythrocytes but does not agglutinate them in either saline or colloid media or after treatment with proteolytic enzymes. Such antibodies can be detected satisfactorily only by the indirect antiglobulin (Coombs) test.

Many suggestions have been put forward to account for the destruction in the body of cells which have been in contact with a non-hæmolytic antibody. These suggestions include an alteration in their osmotic or mechanical fragility, lysis by simple lysins, destruction by enzymes such as β glucuronidase, either intravascular or extravascular phagocytosis, and various other factors such as the alteration of the physico-chemical properties of the cell surface.

Recently, M. Cutbush and P. L. Mollison,[1] using cells tagged with ^{51}Cr and ^{32}P, have studied the relationship between the characteristics of antibodies *in vitro* and the pattern of their destruction *in vivo*. Referring to an earlier study of their own,[2] they point out that when incompatible cells are injected into the circulation, two main patterns of elimination may occur. In one pattern the cells are eliminated with a half-time of less than five minutes and in the other with a half-time of about twenty minutes. In the present study they showed that these patterns could be correlated with the type of antibody involved. For purposes of the study they carried out tests on the following categories: (i) Agglutinating antibodies sometimes associated with hæmolytic activity *in vitro*, such as those of the A B O system and anti-Le[a] and anti-P. (ii) Agglutinating antibodies never associated with hæmolysins *in vitro*, such as anti-c, anti-M, anti-Lu[b], anti-Mi[a] and

[1] *Brit. J. Hæmat.*, 1958, 4:115.
[2] *Lancet*, 1955, 1:1290.

anti-Wr^a. (iii) Antibodies most readily detected by the antiglobulin test. This group could be subdivided further into those, such as the Rh antibodies, which sensitize cells only to anti-gamma globulin serum, and those which sensitize cells to both anti-non-gamma globulin serum and anti-gamma globulin serum. (iv) Red cells sensitized with antibody *in vitro*. Both cells coated with gamma globulin only and cells coated with "non-gamma-globulin-fixing" antibody were used.

It had been shown previously that antibodies which were eliminated with a half-time of less than five minutes were removed mainly in the liver, while those eliminated with a half-time of 20 minutes were removed mainly by the spleen. However, the difference between splenic and hepatic removal may be only quantitative, the spleen removing from the circulation those red cells whose surface properties are so slightly changed that they pass through the liver intact. Cutbush and Mollison found that anti-A and anti-B, which were strongly agglutinating and lytic, or potentially lytic, *in vitro*, produced intravascular hæmolysis, as shown by the presence of radioactivity in the plasma. Less strongly agglutinating antibodies associated with mild hæmolytic activity, strongly agglutinating antibodies not associated with hæmolytic activity *in vitro* and antibodies which sensitize cells to "anti-non-gamma globulin" serum all bring about destruction of red cells predominantly in the liver. Antibodies which sensitize cells only to an anti-gamma globulin serum, and some antibodies which weakly agglutinate red cells at 37° C. and do not bind complement, bring about removal of cells predominately in the spleen.

These experiments throw considerable light on the fate of red cells in the circulation after they have been in contact with different types of antibodies. However, Cutbush and Mollison warn that in the experiments only one-millilitre quantities of tagged cells were injected, and their findings may not apply to larger quantities of incompatible blood entering the circulation. It is to be noted also that only a limited number of antibodies of each type were studied. For example, only two experiments were carried out using cells incompatible with anti-A or anti-B. Furthermore, the results of these experiments suggest that intravascular destruction occurs only when hæmolytic antibodies are involved, whereas, as mentioned above, cases have been reported from time to time in which the antibody is not hæmolytic and yet definite signs of intravascular destruction have been found. The techniques used in this study give some indication of the sites at which cell destruction takes place, but they do not indicate the mechanisms which bring it about, and this fact may limit the use of tracer techniques for studying this problem. Comment on the other methods which have been used by various workers in the study of red cell destruction is beyond the scope of the present discussion, but it seems that much more work is needed, using various methods, before the problem is finally elucidated.

MEDICAL CARE IN CUBA.

A VERY unhappy situation has prevailed for some time in Cuba which has brought the doctors of the country into severe conflict with their Government. The principal issue appears to have revolved around the right of doctors to treat everybody, without respect of persons, and in particular the right not to be penalized for treating those who are openly opposed to the Government. The World Medical Association has taken an active interest in this situation and has done everything in its power to adjust it—with, unfortunately, only limited results.

However, advice has now been received that Dr. Louis H. Bauer, Secretary-General of the World Medical Association, has just returned from a semi-official visit to the Cuban Medical Association (Colegio Mèdico Nacional de Cuba), where he participated in the official opening of the new 22-storey headquarters building. Dr. Bauer reported that, while in Havana, he had sought other sources than the medical profession from which to obtain information about the current status of the medical profession with respect to the rendering of medical care services. He stressed the fact that he had avoided discussing the situation with the doctors, as he did not wish to involve them in any possible danger of retaliation for his observations. It appears that since October, 1957, when the World Medical Association intervened in the interest of humanitarian medical services for all people in Cuba, the situation has improved somewhat; in particular the Cuban Medical Association is now permitted to hold meetings and to publish its journal. However, the medical profession continues to need the support of the World Medical Association in its efforts to provide medical services to anyone needing aid on the basis of caring first for those most in need of those services, regardless of race, colour, creed or partly politics. This is the duty and the right of every doctor under the World Medical Association's Declaration of Geneva and the Red Cross Convention of 1949.

A number of other facts of interest emerge from Dr. Bauer's report. Apparently the revolution in Cuba is active, with actual fighting in all the provinces except Havana and Matanzas. In the eastern provinces, doctors are forbidden to give medical aid to revolutionists. If they do so, they are warned to leave within two hours or be killed. As a result, hundreds of doctors with their families have been forced to seek refuge in Havana, Mexico and the United States. Education is at a complete standstill. Even in Havana, the university and all public schools remain closed, and all the faculty members are unpaid.

Dr. Bauer has appealed to the other 54 member associations of the World Medical Association to implement the provisions of the resolution for the support of the Cuban Medical Association adopted at the XIIth General Assembly in August, 1958, stressing the importance of basing all support and appeals upon the humanitarian precepts of medical service, which can and must be completely divorced from the political aspects of the situation. As he rightly asserts, it is the duty of the members of the World Medical Association to insist that every doctor everywhere in the world be protected in carrying out his humanitarian duties and that his rights in doing so be recognized.

INTERFERENCE WITH WORLD TRAFFIC BY QUARANTINE MEASURES.

AN unusual amount of interference with world traffic has occurred this year because a number of countries have put into operation quarantine measures exceeding the limits laid down by the International Sanitary Regulations. This was reported recently to the Committee in International Quarantine of the World Health Organization. Introduced as a sequel of severe cholera and smallpox epidemics in several Asian countries in the early summer, some of these measures were the most excessive known to WHO since, in 1927, the International Sanitary Regulations came into force. During a recent meeting in Geneva, the Committee in International Quarantine therefore reminded States of their obligations under the Sanitary Regulations and recalled the fact that these sanitary measures are the maximum which a State may apply for protection against quarantinable diseases. The Committee also recalled the fact that the essential aim of the Sanitary Regulations is to ensure the greatest possible security against the international spread of disease with the least possible interference with world traffic. During this year, national health authorities in at least ten countries have violated the International Sanitary Regulations by placing international travellers in quarantine. One country in the Eastern Mediterranean, for example, imposed five days' isolation on all travellers from a neighbouring country. According to the regulations, isolation of travellers is not permitted provided they carry valid vaccination certificates.

Abstracts from Medical Literature.

DERMATOLOGY.

Fatal Metastasizing Erythroplasia.

M. WITHAM, M. GARRETT, A. LEVINE AND A. JARRETT (*Brit. J. Dermat.*, June, 1958) describe a case of fatal metastasizing erythroplasia. The patient presented with numerous psoriasiform patches of Bowen's disease and one large brilliant red plaque-like erythroplastic patch. Histologically, one portion of the patch had the structure of Zoon's type, which he considered non-precancerous, and another portion showed Bowenoid changes in the epidermis. Later, the plaque became partly malignant and spread rapidly as cancer *en cuirasse*. It also gave rise to copious visceral metastases which proved fatal. In the earlier biopsies evidence of sweat-duct proliferation was revealed, and in the later ones features resembling nævus syringadenomatosus papilliferus, except that the duct cells had become malignant and filled the lumen. It is suggested that this is a case of sweat-duct carcinoma with features indicating relationship between nævus syringadenomatosus papilliferus, Paget's disease, both types of erythroplasia and some cases of Bowen's disease.

The Treatment of Syphilis with Antibiotics other than Penicillin.

S. OLANSKY AND W. GARSON (*Arch. Dermat.*, June, 1958) state that numerous antibiotics have been demonstrated to be effective antitreponemal agents in animals. Chlortetracycline, chloramphenicol and possibly oxytetracycline have been successfully used in the treatment of sufficient numbers of patients with syphilis to demonstrate their effectiveness in early syphilis, benign late cutaneous syphilis, neurosyphilis and prenatal syphilis. There is little reason to believe that chlortetracycline, chloramphenicol or oxytetracycline would not be effective in latent syphilis and cardio-vascular syphilis in adequate dosage.

The Use of Amodiaquin ("Camoquin") in the Treatment of Polymorphous Light Eruption.

M. H. CAHN, E. J. LEVY AND B. SHAFFER (*Arch. Dermat.*, August, 1958) have studied nine patients with a history of recurrent prurigo and plaque-like forms of polymorphous light eruption. Amodiaquin was effective in the treatment and suppression of the eruption in all. It successfully suppressed and also prevented an eruption in one patient in whom quinacrine, chloroquine and hydroxychloroquine had been ineffective.

Treatment of Port-Wine Hæmangiomata by Electrodesiccation.

D. E. VANDER PLOEG (*Arch. Dermat.*, June, 1958) discusses the method of treating hypertrophic port-wine hæmangiomata by electrodesiccation. There has been no evidence of keloid formation in the treated patients. Although the cosmetic result is not 100%, the improvement in morale has been remarkable. The author does not treat more than four or five square inches of lesion at one time. He treats a small test area first, to estimate the degree of required improvement, and to observe whether there is a tendency to keloid formation.

Dermatitis of the Fingers from Rosary Beads.

A. RUBIN (*Arch. Dermat.*, June, 1958) describes a case of dermatitis of the fingers from rosary beads. The rash involved the terminal joint section of the palmar-lateral part of the right index finger and the palmar tip of the right thumb. Within a few weeks the patient developed a similar eruption on the same areas of the left hand. The eruption remained more marked on the right hand. The picture was that of contact dermatitis. It was only after she discontinued using her rosary beads that the condition cleared. The dermatitis began shortly after she had begun using a rosary made from olive wood imported from Italy, which was lacquered with plastic.

Planing for Precancerous Skin.

E. EPSTEIN (*Arch. Dermat.*, June, 1958) sent a *questionnaire* on the subject of planing of precancerous skin to 120 dermatologists who were known to employ the procedure in their practice. The precancerous conditions subjected to planing were actinic or senile skin, radiodermatitis, leucoplakia, xeroderma pigmentosa and epitheliomata, especially superficial basal celled carcinomata. Apparently planing is more beneficial in squamous celled than in nodular, cystic or ulcerated basal celled carcinomata. Most epitheliomata can be treated by dermabrasion. However, this is not the treatment of choice for such tumours. Probably any accessible surface can be planed if planing is therapeutically indicated. The use of planing in precancerous skin is increasing. It may replace the use of this modality in the treatment of scars, which is at present the prime indication for planing.

Fungous Disease as a Complication of Steroid Therapy.

R. E. BURNS (*Arch. Dermat.*, June, 1958) states that an awareness of the risk of dissemination of fungous disease by steroid therapy is necessary. A complete examination of the patient, including X-ray examination of the chest, must be performed before major steroid therapy is initiated. If this is done, and if reasonably thorough follow-up examinations are performed, there will be little chance of serious trouble. Despite these precautions, an occasional patient will develop systemic fungous disease. Superficial mycotic infections should always be brought under control before the start of treatment if it is feasible, and if not, active treatment should be carried on during systemic steroid therapy.

The Nylon Brush.

A. SAVILL (*Brit. J. Dermat.*, August-September, 1958) has found that modern nylon bristles have very different points: (i) rounded points, like the head of a match—safe to use; (ii) square points, which look as if square-cut—not safe; (iii) square-cut ends, gently rounded off—not so harmful. Loss of hair can often be attributed to the use of a nylon brush. Several cases are quoted. As the old-fashioned hair brush is becoming rarer, so cases of the type quoted will become more common. In any hair loss, the type of bristles of the nylon hair brush should be examined under the low power of the microscope.

Exfoliative Dermatitis Complicating Chloroquine Therapy in Rheumatoid Arthritis.

L. W. GRANIRER (*Arch. Dermat.*, June, 1958) reports a serious type of reaction to chloroquine manifested by severe purpura, œdema, erythema and exfoliative dermatitis. There was no previous history of dermatitis, allergy or drug sensitivity. Symptoms occurred 20 days after the beginning of treatment with a single dose of 250 milligrammes of chloroquine per day. Patients should be warned of possible severe skin reactions.

Epitheliomata Treated by Dermabrasion.

C. M. RIDLEY (*Brit. J. Dermat.*, August-September, 1958) has treated a small series of patients with epitheliomata by dermabrasion. No patient has been followed for much more than a year. The author considers dermabrasion worth a trial, particularly for elderly patients, for patients with multiple lesions, and when there are difficulties in the way of excision or radiotherapy. It is simple, safe and free of unpleasant sequelæ, and causes very little inconvenience to the patient. At best it may result in a cure, and it does not make further treatment, if this is ultimately needed, any more difficult. Careful supervision of the patients afterwards is essential.

UROLOGY.

Carcinoma of the Prostate.

T. W. MIMPRISS AND A. F. FERGUSON (*Brit. J. Urol.*, March, 1958) have reviewed 123 cases of carcinoma of the prostate, with a five-year follow-up of 36 cases and a three-year follow-up of a further 40 cases. In a very large proportion of sufferers from this disorder it is the disease itself which is the actual cause of death when death occurs. Those whose death is due to other causes have all been shown to have carcinoma still present in the prostate. Radical removal of the carcinomatous gland is the only possibility of complete cure, but in the authors' experience this is hardly ever applicable. It is in favour in the U.S.A., but not so in Britain, for the simple reason that so few patients are seen in whom it can be contemplated. The higher proportion of early cases seen in America is attributed to frequent routine examinations. Palliation by endoscopic resection and the hormonal attack gives increased life expectancy, but these measures never seem to cure the disease. It seems that survival depends more on the natural course of the disease than on any other factor, and that, on the average, the effect of hormonal treatment is to prolong life by about 18 months. In this series the percentage survival of patients without

metastases when first seen was 45% at three years and only 17% at five years. In the group first seen after metastases had appeared, the percentage of survival at three years was 30% and at five years 20%. The latter figure is, however, not statistically significant, since the number of cases here was very small. In this series the hormonal attack was by giving œstrogens; orchidectomy was not done.

Impotence and the Leriche Syndrome.

V. J. O'CONOR (*J. Urol.*, September, 1958) states that sexual impotence often results from thrombotic occlusion of the terminal aorta. This symptom is an integral component of the complex commonly referred to as the Leriche syndrome. Furthermore, the impotence may be the earliest complaint, antedating symptomatic and clinical evidence of peripheral arterial insufficiency. Being aware of this symptom as a manifestation of occlusive arterial disease, the urologist may be in a position to make an early diagnosis, and to institute surgical treatment before irreversible damage occurs. A case is described of a man, aged 35 years, with a five months' history of progressive inability to maintain erection. The ability to produce an erection is dependent on an adequate blood flow in the arteries supplying the corpora and the competency of their innervation. The ability to sustain an erection is more likely dependent on the arterial and nervous supply of the penile musculature (over the bulb and crura). Contraction of these muscles partly occludes the venous return. Under diminished pelvic blood supply, there may be enough arterial inflow to initiate the erection, but insufficient blood supply to the bulbocavernosus and ischio-cavernosus muscles to help them maintain sufficient contraction. In the case reported here, the young man got complete return of erectile function following aorto-iliac thrombo-endarterectomy. Careful study of the vascular system is advised in all patients with impotence.

Sling Operation for Urinary Incontinence in the Male.

C. W. UHLE AND C. H. BRADLEY (*J. Urol.*, August, 1958) report on eight cases in which sling operations were performed for urinary incontinence in the male. The double sling came from the anterior sheath of the rectus abdominis in six cases; fascia lata was substituted in three cases; both procedures were performed in one case. The course of the fascial strips is anterior to the pubis, and they run down to the perineum in fatty tissue, passing up again to be sutured to the surface of the sheath of the opposite rectus muscle. The objective is that the sling should compress the very deepest (proximal) part of the bulb; therefore an open tunnel made by finger dissection is inadmissible, since the sling can move forward and compress the urethra too distally. The degree of tightening of the slings is determined by a special hydrostatic technique during the operation. In spite of all technical care, the results have been disappointing, there being only partial success in four cases. There were many complications, some necessitating surgical intervention for their correction. Final judgement of the value of this operation should not be made until series of cases are reported by various surgeons.

Transition of Cystitis Glandularis to Primary Adenocarcinoma.

J. L. SHAW, G. J. GISLASON AND J. E. IMBRIGLIA (*J. Urol.*, May, 1958) state that there is a close relationship between glandular cystitis (a common form of proliferative cystitis) and primary mucus-secreting adenocarcinoma of the bladder. This relationship has been emphasized in the past, but only on presumptive evidence based on the high incidence of the coexistence of these lesions and similarities of theories of pathogenesis. The main theories are: (*a*) that embryonic rests are the basis; (*b*) the anatomical theory; (*c*) the theory of metaplasia of transitional to glandular epithelium. The last-mentioned is the most widely accepted. The patient whose case is described was followed carefully over a period of five and a half years, and a definite transition from glandular cystitis to adenocarcinoma was demonstrated. This is the first case in which such change has actually been observed and demonstrated. This report adds to the evidence that cystitis glandularis, as well as other forms of proliferative cystitis, is a premalignant vesical lesion. This fact should be kept in mind in the clinical management of patients with these chronic forms of cystitis.

Serum Mucoproteins in Prostatic Carcinoma.

J. A. ARCADI (*J. Urol.*, September, 1958) states that for over 25 years it has been known that there is frequently an increase in the protein-bound carbohydrate of blood serum in patients with carcinoma. In 1949 Gersh and Catchpole presented evidence that the carbohydrate-containing protein of the serum arose from the "ground substance" of the connective tissue. This "ground substance" is the non-fibrillar component, or intercellular cementing substance, through which collagen and reticulin grow, and upon which cells rest (i.e. the basement membrane). Around growing tumours the protein-bound carbohydrate (muco-protein) was found to be extremely water-soluble; it is relatively water-insoluble in normal tissue. It was found by Catchpole in mice that where a large amount of soluble tissue carbohydrate-protein complex existed around the tumour, the serum muco-protein level was also increased. Catchpole concluded that such findings supported the thesis that increased serum glyco-protein (muco-protein) levels arise from ground substance at the site of an invasive growth by a process of depolymerization, whereby smaller water-soluble and diffusible glyco-protein moieties are produced. The author states that approximately three years ago he presented evidence clearly showing that the ground substance of untreated prostatic carcinoma was soluble in a buffer solution at pH 7·0. It was much less soluble when the neoplasm had been treated by castration or œstrogens. Such evidence suggested that the serum muco-protein level might be elevated in untreated prostatic cancer, and decreased after treatment. Two groups of patients were studied; there was a control group of 25 men, mostly in the prostatic age group, but without any sign at all of prostatic carcinoma. The study group consisted of 33 patients with proved prostatic carcinoma; only five of these patients had no demonstrable metastases, although it was apparent, from rectal examination, that the neoplasm had extended outside the prostate. The author shows that the serum muco-protein level is increased in untreated prostatic carcinoma; after treatment the serum level sinks to within the normal range. However, to evaluate this "test" properly, the determinations should be made on a group of patients in whom the tumour is confined to the prostate gland itself. Nevertheless, at this stage the method can be suggested as a further diagnostic test, and may be useful prognostically.

SURGERY.

The Gastro-Œsophageal Closing Mechanism.

G. S. MULLER BOTHA (*Brit. J. Surg.*, May, 1958) discusses mucosal folds at the cardia as a component of the gastro-œsophageal closing mechanism. In 1815 Megendie suggested that mucosal folds occlude the cardia. Since then all references to mucosal folds appear vague, and so the author set out to study the structure and function of these folds in animals and man. Specimens from 30 adults and 115 infants and children were studied by macroscopic dissection and histological examinations. The findings were extremely variable, but did suggest that folds are present during life and disappear after death. Twelve patients who were subjected to partial gastrectomy for benign ulceration were examined by digital palpation. There was evidence of occluding mucosal folds in every patient, although no two of them presented the same pattern. The mucosal fold pattern was similarly studied in 21 different species. All investigations suggested that the physiological mucosal folds at the cardia both in man and in animals are in a state of positive tone and constant activity which are mainly influenced by the surrounding musculature. However, the experimental observations also favoured a definite intrinsic force in the folds themselves, quite independent of the stomach wall. It was thought that the muscularis mucosæ is specially adapted to control and maintain independent tone in the folds that guard the gastro-œsophageal orifice. The mucosal folds are actively drawn together by the internal œsophageal sphincter to produce a water-tight seal at the cardia. These two factors, the folds and the sphincters, act together in perfectly balanced harmony to form the closing mechanism between stomach and œsophagus. The diaphragm is an important accessory mechanism and maintains the normal position of the gastro-œsophageal segment, so that the closing mechanism can act with the greatest advantage. The angle is an organ characteristic which, as such, plays no part in the closing mechanism. A mechanical valve does not exist.

Special Article.

REHABILITATION: V. MANAGEMENT OF THE DISABLED IN THE HOME.

THE family doctor is the person who most frequently encounters the problems presented by the disabled in the home, and who often has felt frustrated and sad because there appeared to be no future for the victims of strokes, paraplegia and chronic nervous diseases and for the badly crippled arthritic, as well as for the victims of serious trauma. Today, with modern techniques in rehabilitation, this outlook has changed, and the horizon of the disabled person has extended far beyond its previous limits. Overseas it is now accepted by all patients in hospitals possessing a rehabilitation centre that their home situation will be thoroughly reviewed before they return to it; and they look forward to their discharge from hospital. Up to date in Australia we are breaking new ground, but nevertheless making satisfactory progress. We should like to pass on our methods and offer help to any member of the medical profession who encounters these difficult problems.

With the trends of modern living, it has become very difficult to find people who are financially able to care for the chronically disabled at home. Many reasons are given; e.g., all members of the family are working and cannot meet their financial commitments if they cease; or the space available in small modern homes is not suitable for the care of these people; and the sense of duty towards parents and unfortunate relatives seems to have changed. Granted there are still many devoted daughters whose lives are sacrificed, and many family doctors whose visits are a breath of life to the housebound lonely soul; but there are also many others who take no interest whatever in this type of work, and many of the younger generation have come to regard all invalids as a hospital problem. It is for the average situation that our methods are described.

There comes a stage in hospital when the chronically disabled patient asks continually what will be his future, and where will he spend it. Thousands of hospital beds required for the acutely ill are occupied throughout the world by those suffering from chronic illness, either because there is no other suitable accommodation or because there is no money to pay for it, and the patient remains in the ward where his acute illness began. Highland View Hospital, Cleveland, Ohio, U.S.A., is a large hospital devoted to the care of chronic illness. In 1956 there were 515 new admissions to that hospital, and in the same period 327 patients were discharged to the community, 193 of these to their own homes and the others to institutions; the hospital has a bed capacity of 550. For many years the turnover of beds in that institution was almost nil, but a properly organized team approach has achieved this very desirable result. That particular institution accepts very seriously disabled people with all types of difficult and long-term problems; there they have successfully found means of helping the family to adapt the home physically and its atmosphere psychologically to the needs of the disabled person, and have set a useful pattern for other institutions to follow.

Methods.

The services of a trained almoner are extremely helpful, but in country areas in Australia the thoughtful family doctor automatically accepts these tasks and does them very well. Some new ideas to help in these situations are offered. There is a great advantage in having the personal care and knowledge of one person dealing with the situation from first to last.

In large city institutions where the resident staff is constantly changing, the almoner deals with these problems, and upon her care and interest depends the bed turnover of the hospital. Whenever possible, discharge arrangements are discussed before the patient is admitted; relatives and friends are told that the acute phase of the illness will receive attention in the large institutions, but that there will be a change as soon as some progress is shown by the patient. The team approach allows pre-admission, progress, discharge and follow-up conferences on every patient; this is the ideal method, and allows short and long term goals to be arranged. The patient is constantly aware that arrangements for his future are under consideration, and this allays the anxiety existing in many instances and preventing progress.

Where is the patient to go when the period in hospital is complete? Undoubtedly the home, if satisfactory, is the best place. If suitable arrangements and adjustments cannot be made, then a convalescent home is often the only answer; frequently the type and location of this are determined by the patient's financial position.

Some responsible person should visit these small hospitals and make careful inquiries about the routine adopted and the method of control of the hospital. Custom dies hard, and elderly sick people dislike serious changes in life-long habits. Many of these hospitals refuse fluid after 4 p.m. because they do not provide regular night staff and aim to avoid the need for bedpans during the night; also one finds that the patients are confined to their beds and rooms a great deal of the time, with no physical exercise and no occupation for their minds. According to modern standards this is wrong both in the acute and the chronic hospital. A busy patient is a happy patient; while the bed is a constant focus of attention, no thought is given to recovery. Every patient in a convalescent hospital should be fully dressed each day (corsets and shoes included), encouraged to take an interest in her personal appearance, and taken if possible into the sunshine and fresh air for a walk. If this is not possible, she should be taken into a sunroom or lounge. Meals should be eaten at a properly prepared table, not on trays balanced on shaky knees, and a home situation with tender loving care should be attempted continually.

In the acute hospital, with early ambulation today it is possible to provide a change of scene for patients continually. At Highland View Hospital they instituted a sunroom programme to encourage the convalescent patients to do useful things and work with other people. The patients spend each afternoon in groups on a sun veranda, sitting either in comfortable chairs or even in their own beds, doing useful tasks for the hospital—they fold linen, do mending, have occupational therapy, listen to the radio, watch television, have afternoon tea and chatter, returning to the wards mentally and physically refreshed; this is not impossible in any hospital. Relatives are encouraged to visit the hospital and help at these sessions, which are supervised by an occupational therapist; there they are also instructed in the care of the patients in the wards; bathing, feeding, dressing and assisting them in their physical exercise routine, they gain confidence and feel that the home situation is not impossible.

When the patients are able to return to their own homes, the almoner visits the home and evaluates it as a home as well as a house. The country doctor already knows these facts from his long association with the family; but there are many simple adjustments which can be made to allow the invalid to return home and to live safely.

When the social situation is being assessed, it is necessary to know of the patient's place in the family unit before his illness, the degree of family unity and stability, the family's financial position and its desire to take the invalid back and care for him. The security afforded by knowing that problems arising can be discussed with the almoner or doctor at any time gives many people courage to attempt home care of the invalid. Careful planning on the spot with the relatives gains their confidence, and the assurance that if the task proves too great a burden help will be given frees many a much-needed hospital bed. In order to fit into the home situation as satisfactorily as possible, the invalid should be made adequate in his acts of daily living, and as far as is possible made to feel that he can be an asset, not a nuisance, in the home. The approach begins in the early acute stage of illness by the nursing staff, who encourage him to help himself whenever possible, giving due praise for any small achievement. Relatives are impressed with the idea that their task will be easier in the future if they are not over-protective, and although it may be slower to allow the patient to help himself, in the end his independence will pay dividends. With the present forty-hour week in nursing, neither the patients nor the nurses know each other very well; but an understanding ward sister is the key person in this routine. She teaches her staff constantly to build up a future for the patient, and she finds occupations for him. Sets of simple routine physical exercises to be done at stated intervals can be carried out whether a physiotherapist is available or not. There should be a constant daily routine as well as washing, feeding and bed-making, to turn the mind towards a useful future.

To avoid a heavy burden on the shoulders of one person charged with the care of the invalid in the home, if a regular roster of relatives and friends is organized to undertake shopping, letter-writing, telephoning, care of clothing, adapting furniture and utensils, arranging transport for medical and dental care and supervision etc., this brings fresh interests into the patient's life and distributes the tasks. Many people wish to help, but hesitate to do so unless special tasks are allotted to them.

Financial assistance from social service and other agencies should be discussed and arranged. A list of all agencies is available to any doctor who requires it from the New South Wales Council of Social Services, 33 Macquarie Place, Sydney. Many people are unaware of the benefits made available to them both by the State and by Commonwealth agencies.

With this brief outline of the social and psychological approach, it will be realized that successful lasting return of the invalid to the home or convalescent hospital cannot just happen—it must be carefully planned, and the full cooperation of patient, hospital staff, relatives and friends obtained; in other words, their attitude of mind has to be changed to accept the individual problems arising.

It is well to remember that every patient is entitled to a private consultation with his doctor if he wishes. For this reason, visits to the surgery even by ambulance should be encouraged as affording a change of scene and a possibility of discussing personal problems. The brief round of the doctor in a convalescent hospital allows little time for assessment of the patient's diet and fluid requirements and his satisfaction or otherwise with the hospital.

General Principles of House Adaptation.

These include the means of transport to and from the home, the approach to the house and every room in it. An assessment is made of the patient's ability to perform his acts of daily living, the home is assessed against these, and the necessary adjustments are designed and gadgets made if necessary to suit the individual problems.

Wheel-chair techniques require a separate section.

Transport.

If possible, before leaving hospital the patient is taught step climbing, road crossing, and how to get in and out of public transport. When a motor-car is driven, it is possible to have the necessary adjustments made and a driver's licence endorsed accordingly. If the patient is confined to a wheel-chair, transfer techniques are learnt and arrangements are made for his own chair; modern wheel-chairs are scientifically constructed with good brakes and adapted to the patient's disability. Doors may have to be altered to admit a wheel-chair. If crutches are necessary, crutch walking is taught well before discharge from hospital, and the patient has general muscle-conditioning and balance exercises and is taught to walk between bars before attempting to walk on crutches; he is also taught how to fall and to get up, as well as how to climb steps. The use of Canadian elbow crutches is encouraged whenever possible.

The Approach to the House.

When steps are steep or dangerous, a ramp must be built and a stable grab rail constructed. It is important to extend this rail beyond the level surface at the top of the stairs and also a short distance beyond the bottom step. Security and efficiency in entering the house encourage the patient to go out unattended and to mix with other people—a very important psychological factor.

The Hall.

If the patient is confined to a wheel-chair, steps should be taken to obtain a wheel-chair to fit the door. If this state of affairs is to be permanent, removal of the architrave and slight enlargement of the door will frequently give the extra few inches required. Sometimes swinging a door outwards will help. The minimum of basic alterations helps the patient to feel less of a burden.

The Bedroom.

For the invalid this is very important—there should be a minimum amount of necessary furniture, but it should all be functional.

Castors should be removed from the bed to ensure stability. If necessary, the bed should be pushed into the corner, care being taken that the patient's eyes are shielded from bright light if it is opposite a window. If it is necessary to adjust the bed to a wheel-chair or a particular height, small blocks can be made for the legs or put under the four corners of the mattress; also where a wheel-chair is required, space should be allowed so that it can be placed parallel to the bed facing the head with the side, so that the involved extremities are close to the bed.

Clothes should be arranged where they are easily accessible, and a small trolly with necessary cosmetics and other personal belongings will save much fetching and carrying.

Back rest and foot boards to relieve the weight of bed clothes should always be available, and all covers should be warm and light.

Clothing should be carefully chosen and simple in type, whenever possible made of crease-resistant material easily washed—e.g., seersucker, nylon and drip-dry cottons. Pyjama jackets, shirts, nightgowns and dresses should be opened completely down the front. Cuffs of men's shirts should be buttoned at the wrist on the affected side before the shirt is put on; elastic thread will often serve a useful purpose in securing a cuff and allowing expansion. Always put in the affected arm first—remove the shirt in the opposite order. A clip-on bow tie or tie with elastic stitched to the bow on either side will also help. Trousers should be at least one size larger than usually worn, for easy dressing; these should be put on the affected leg first. Zipper fastenings instead of buttons are useful. For paraplegic females, slacks with zippers on the outside of both legs from cuff to waist are very comfortable.

Shoes should be comfortable and easily put on. A long-handled shoe-horn is helpful, and also elastic laces, now obtainable in this country, save lacing and unlacing.

Stockings present a problem to the patient with stiff knees; there are several gadgets which can be made to help put them on. Tape loops sewn on the tops of stockings assist in pulling them up more easily.

The Bathroom.

Before disorganizing the family routine in the bathroom, it is better to try the patient in his own home surroundings before making extra devices. If the patient cannot manage, the following additions are found useful: grab bars on each side of the toilet; an elevated toilet seat, to be placed over the toilet and hinged back out of the way of other people. (This can be achieved by light blocks attached to a second toilet seat to fit over the existing one. The height is determined by the individual disability and the patient's height.)

Bathing can be achieved by various means; there are elaborate seats, but these are not necessary A rubber bath-mat should be placed in the bottom of the bath; a chair with the back removed or a stool standing on the bath-mat can be used. If the patient is in a wheel-chair, these should be arranged at the appropriate height. A very useful rubber hose is available; at one end it can be attached to both hot and cold taps, and at the other end delivers water of the correct temperature. If it is preferred, a shower may be taken with the patient seated; but all hot pipes should be protected and the temperature of the water watched. Long-handled bath sponges with a place for the soap will allow the patient to reach almost any part of the body. Suction cups attached to nail brushes are available, and will help the one-handed patient to keep his nails clean; a second brush of this type is useful for dentures. A towelling bath mitten with soap in a pocket of it is most helpful. For the woman, a long lipstick holder and a nail file fixed in a stationary position are also helpful. An electric razor is very helpful for a man, and mirrors should if possible be tilted or arranged conveniently; if not, a mirror should be dipped to wheel-chair height or specially placed. Toilet articles in plastic containers are easier to handle, especially those with sprays and packed under pressure.

The Kitchen.

This subject alone should occupy a separate article, but there are certain basic principles that should be incorporated in any kitchen—namely, the physical and mental limitations of the patient must be carefully considered before she is subjected to the activities of the kitchen. Step-saving techniques are important, with grouping of commonly used articles to save unnecessary movement. There should be no cupboards under the sink, to make room for the wheel-chair. An overhead sling should be provided to support paralysed arms while working. All kitchen furniture should be at a suitable height for the particular patient. Extensions on taps, specially adapted gadgets for disabled hands and a strongly constructed trolly for transport of china, pots and pans, etc., help the disabled to play their part in useful management of the home. Always remember that the patient who disliked housework before illness does not suddenly become an enthusiast about it.

Cleaning is aided by long-handled mops, brooms and dust-pans, and an apron with a very large pocket to hold dusters, brushes, cleaning materials, etc., saves many unnecessary steps.

Ironing can be done by the patient sitting on a comfortable stool of correct height; featherweight irons are useful, and one of our most helpful additions is an adjustable ironing board.

The Living Room.

The following are important points relating to the living room.

1. Scatter rugs should be removed, and carpeting and fringe, if present, tacked down.

2. Highly polished floors should be eliminated; they are unsafe even for the able-bodied.

3. Furniture which obstructs the doorways, making the passage of wheel-chair or crutches or making walking difficult, should be rearranged.

4. Extensions of light cords for easy accessibility from wheel-chair level or for patients with other disabilities are recommended.

5. Commonly used furnishings, such as radio, television set and telephone, should be readily accessible.

6. A sturdy and comfortable upholstered chair with good padded arm rests should be available to the patient; if the wheel-chair is used, it should correspond to the same height. Castors should be removed from the chair for stability.

7. Lighting, whether natural or artificial, should be adjusted, so that it does not shine directly in the patient's face.

8. Good ventilation is essential if the patient is to spend many hours in the room.

9. Warmth from a light knee rug will often allow the patient to remain without a fire until late in the day.

It is advisable to keep the adjustments to a minimum, to save the invalid embarrassment and the family annoyance.

Conclusion.

Every problem is an individual one, and must be solved by special planning. Mass-produced equipment is seldom as useful as simple adjustments designed for individual needs.

In other countries there is a government service to provide home adjustments, wheel-chairs and home help; it is considered very important to keep the family unit intact and happy, and to reserve hospital beds for the acutely ill and for accident patients. The Royal South Sydney Hospital Rehabilitation Centre has instituted this home service for its patients, and is willing and happy to give advice at any time to anyone requiring it.

Summary.

1. Prepare the patient and relatives for early discharge from hospital by free discussion of the problem from the time of admission and by careful planning and preparation.

2. Assess the home situation as a home and a house related to the patient's pre-illness status.

3. Take each area of the home, including transport and approach, and evaluate and deal with the problems.

4. Give a hopeful outlook for the future, planning for the patient a useful place in the home as well as in the community.

M. NAOMI WING,
Sydney.

Out of the Past.

In this column will be published from time to time extracts, taken from medical journals, newspapers, official and historical records, diaries and so on, dealing with events connected with the early medical history of Australia.

THE SYDNEY BENEVOLENT ASYLUM.[1]

[From the *Australasian Medical Gazette*, November, 1882.]

A CIRCULAR has been issued inviting medical men to send poor women, suffering from diseases peculiar to their sex, to the department allotted to such cases in the Sydney Benevolent Asylum. It is to be regretted that the medical arrangements in this Institution are still such as not to commend the confidence of either the profession or the public. The profession apparently think the department a joke, and such it is likely to remain until the consulting staff is organized on a basis suited to the responsibilities of its difficult functions. What other class of men in the community would be expected to undertake duties involving such serious obligations from mere motives of charity? The expenditure of a small fraction of the annual savings of the Institution would enable the Board of Directors to secure the services of two of the best professional men in Sydney as consulting medical officers. Why is this not done?

[1] From the original in the Mitchell Library, Sydney.

Correspondence.

MEDICINE IN CHINA.

SIR: "Current Comment" on "Medicine in China" (M. J. AUSTRALIA, November 15, 1958) notes the resurgence of traditional Chinese medicine, and states that "this curious development is hard to understand in any country, Communist or otherwise".

I think, however, that the reason for it is quite simple. There is now occurring in China a tremendous renaissance of and emphasis on everything indigenous. So we find great interest and development taking place in opera and drama, arts and crafts, literature, archæology, folk-lore, minority groups and the rest. Chinese medicine shares in these revivals, not because it is better than any other system, but simply because it is Chinese. It is part of present-day Chinese chauvinism, which is the full swing of the pendulum from the violent Reform Movement of 1898.

Perhaps it ought to be encouraged even more—the population increase in China is horrifying.

Yours, etc.,

147 Wollombi Road,
Cessnock,
N.S.W.
November 17, 1958.

HENRY W. H. MCCLELLAND.

CARDIAC ARREST: A PLAN OF ACTION.

SIR: I should like to congratulate Dr. Bernard, albeit somewhat belatedly, upon an excellent plan of action when confronted with a case of cardiac arrest. There are, however, certain points in this paper which I feel require comment.

She mentions that an attempt should be made to restart the heart by a "quick right auricular puncture". I consider this is a waste of time and should never be used.

If the condition of cardiac arrest is suspected, one should go ahead with massage, and as Milstein[1] mentions in his very complete paper on the subject, the criterion of whether to commence or not should be presence or absence of the carotid pulses. Absent carotid pulses, when arrest is suspected, should be the signal for thoracotomy.

Dr. Bernard in her paper describes a method of cardiac massage which is manifestly impossible if the pericardium has not been opened—namely, compression of the heart by the whole hand against the sternum. This method, even when the pericardium is opened, causes quite severe distortion around the base of the heart, which in turn interferes with cardiac filling.

I believe that once the initial incision has been made and the hand inserted, if there is not a response to finger and thumb compression within, say, a dozen squeezes, no time should be lost in incising the pericardium, cutting two costal cartilages, and squeezing the heart between the palms of both hands.

A mechanical retractor, if available, is a great help here, but not if an assistant can hold the ribs apart very adequately with his fingers. Without some sort of retraction, the hands become very cramped within a few minutes. Even someone who has not done this procedure before should be able to reach this stage of the proceedings within some ninety seconds or so after the skin incision. I was glad to see that the futile procedure of massage through the intact diaphragm was not mentioned.

[1] *Ann. Roy. Coll. Surgeons England*, 1956, 19: 69.

Now, regarding the letter of Dr. Reeve in the issue of November 8, 1958, he mentions that a pressure below 60 millimetres of mercury is inadequate to maintain cerebral circulation. Certainly a pressure as low as this is undesirable; but every surgeon of experience has seen exsanguinated patients who have had a pressure below this level for a considerable period of time, i.e., 15 minutes or more, and yet have responded to resuscitation and have recovered with no evidence of cerebral damage. In other words, a low pressure which is maintained is better than no pressure.

Finally, the method of using the fingers and thumb, which is mentioned by Dr. Reeve, is all right if it is used gently and initially before a complete exposure, as mentioned above, is attained. But in a case requiring prolonged massage it is extremely dangerous, as, particularly in children, the thumb may perforate the ventricle—a most embarrassing accident, but one that has been treated successfully by suture.

In conclusion, the widest dissemination of these techniques should be given, as any doctor may be faced with this problem, and it can occur not only in the operating theatre, but in one's consulting room while doing such simple procedures as bronchography or the tapping of a hydrocele.

Yours, etc.,

C. F. A. CUMMINS.

The Rockhampton Clinic,
166 Alma Street,
Rockhampton,
Queensland.
November 12, 1958.

NOMENCLATURE IN ANÆSTHESIA.

SIR: In answer to Dr. C. J. B. Armstrong (M. J. AUSTRALIA, November 22, 1958) *re* the confusing nomenclature of the muscle relaxants, it would appear that the true pharmacological action of these drugs is not appreciated.

"Tubarine" does not act directly on the myo-neural junction, but competes with the normally produced acetylcholine for a position on the receptor sites in the junction, thereby preventing the depolarization of the end-plate membrane, which would occur if acetylcholine were allowed to act unhampered. "Tubarine", therefore, is not a polarizer, but a preventer of depolarization, and is classified as a competitive or non-depolarizing muscle relaxant.

"Neostigmin" acts by preventing the action of cholinesterase, thereby allowing acetylcholine produced to reach levels sufficient to overcome the effects of "Tubarine". It is, therefore, not itself a depolarizer, but allows acetylcholine to carry out its normal function of depolarization.

With regard to the muscle relaxation exhibited by the other groups of relaxants—e.g., decamethonium and succinylcholine—the mechanism involved is not similar, but opposite, to that of the competitive class. These drugs, by a direct action on the myo-neural junction, cause a change in permeability in the end-plate membrane to sodium and potassium, with a subsequent depolarization of the membrane. Such drugs are rightly called depolarizing muscle relaxants, and their effects are enhanced, not diminished, by anticholinesterases.

Yours, etc.,

G. J. DALGARNO.

16 Belmore Street,
Burwood,
N.S.W.
November 24, 1958.

STREPTOMYCIN POISONING, RENAL DISEASE AND THE ARTIFICIAL KIDNEY.

SIR: We have been concerned by the number of patients we have recently seen suffering from the toxic effects of streptomycin given in the presence of renal disease. We should like to remind readers of the dangers of this combination, and to mention the part which treatment with an artificial kidney can play in certain circumstances.

Streptomycin is normally excreted in the urine. If renal function is in any way subnormal, then excretion of the drug will be impaired, and its retention within the body is liable to cause toxic effects. These most commonly involve damage to the vestibular nerve leading to disturbances of balance, which are usually permanent, extremely troublesome, and may be completely disabling. Hearing may also be impaired. As ordinary dosages can cause this in the presence of relatively minor degrees of renal dysfunction, there should be some assessment of renal function—a check on urinary volume, maximum attainable specific gravity, proteinuria, and perhaps a measure of blood urea—before giving streptomycin. But when there is gross renal dysfunction, then even small amounts of the drug may cause dire effects. We have seen this occur after only 1·5 grammes. Especially when there is oliguria, the drug is best avoided.

In this regard, we have been alarmed by the number of patients referred to us with acute oliguric renal failure and to whom streptomycin has been given. Of 40 patients with this disease treated by our unit in the past two years, 13 have been given streptomycin during the period of oliguria, and several of them are now permanently incapacitated. We have shown that streptomycin can be removed from the body by treatment with an artificial kidney, and we now believe that streptomycin given to a patient with anuria, or oliguria, constitutes an absolute indication for dialysis with this machine. Treatment should be given as early as possible, before toxic effects have appeared. Only one of our seven patients so treated within 10 days of the onset of oliguric renal failure has suffered damage from streptomycin, and she is not severely handicapped.

However, avoidance of the drug is better than attempted removal. Streptomycin is rarely absolutely necessary. Paying some attention to the state of the kidneys before, or even after, giving the drug will reduce the incidence of this disabling complication of therapy.

Yours, etc.,

W. E. L. DAVIES,
K. D. G. EDWARDS,
H. M. WHYTE.

Clinical Research Department,
Kanematsu Institute,
Sydney Hospital,
Sydney.
November 21, 1958.

CORONARY HEART DISEASE IN YOUNG ADULTS.

SIR: The recent editorial in THE MEDICAL JOURNAL OF AUSTRALIA of November 15, 1958, mentioned yet another suggested ætiological factor in the coronary heart disease with which we know Australia is as badly affected as America.

There have been many recent articles, particularly in American literature, concerning studies on the sera of normal and abnormal patients, with reports on such factors as cholesterol-phospholipid ratio, alpha-beta lipoprotein-cholesterol ratio, pre-beta lipoprotein band, etc., only serving to complicate the issue.

Reports of treatment have been no more helpful; cholesterol levels can be lowered, and ratios can be altered by substances varying from unsaturated fatty acids to œstrogens, each author having his favourite combination, but rarely being able to make a positive recommendation for general use.[1] This is reflected by the slowness of the drug companies to place any preparation on the market. In N.S.W. only one (so-called) anti-arteriosclerotic substance is available to the patient; it is expensive and appears to have had insufficient clinical trial to justify its use.

To illustrate one point of practical importance not generally realized, a series of cases studied recently at the Royal Prince Alfred Hospital is mentioned below. Between February and November, 1958, there were nine males under 45 and two females under the age of 50 admitted to this hospital suffering from myocardial infarction or coronary insufficiency, unassociated with hypertension. The family histories of all these patients were studied, and where possible cholesterol and lipoprotein patterns performed on the serum of their siblings. In three cases a positive finding was recorded. (i) The 42-year-old brother of a male of 38 with proven infarction has severe peripheral vascular disease with a normal cholesterol. (ii) The otherwise normal 40-year-old brother of a 41-year-old male with anterolateral infarction was found to have a serum cholesterol of 420 milligrammes per 100 millilitres (this level has been reduced to 280 by treatment with a low fat cholesterol diet and sunflower seed oil). The original patient himself had a normal cholesterol level. (iii) The 42-year-old brother of a 45-year-old male with coronary insufficiency was found to have signs of aortic stenosis, but left ventricular puncture revealed no gradient across the aortic valve, and the cause

[1] *Circulation*, 1958, Volume 18, page 432.

of his markedly enlarged left ventricle remains undetermined, but could be coronary arteriosclerosis. These cases emphasize the importance of careful family investigations in this group of patients.

By following this family line of study outside the age group mentioned, two family trees with hypercholesterolæmia were discovered. In one of these only the males were affected, the youngest, aged 19 years, having a serum cholesterol of 390 milligrammes per 100 millilitres.

In the treatment of the young patient with coronary disease—and the word "young" is of prime importance—it seems reasonable to suggest the following principles:

1. Long-term anticoagulation where practicable appears well justified in myocardial infarction or coronary insufficiency, and there is little doubt that this is not being practised enough.

2. An elevated cholesterol should be attacked vigorously with the combined administration of low cholesterol-fat diet and some form of unsaturated fatty acid preparation, neither apparently being effective alone. At the moment, although no unsaturated fatty acid mixture is available for other than research purposes, it is hoped that it soon will be, and in an inexpensive form. The danger here, of course, is that it will be used as a general cure for all forms of arteriosclerosis, irrespective of age or serum cholesterol level.

3. In a patient with a normal serum cholesterol, it does not appear justified to commence a treatment which is of doubtful value, of definite inconvenience to the patient (and his wife!), and which is very difficult to terminate once started.

In particular, products meant to change the cholesterol distribution ratio seem an unnecessary expense and of little more than placebo value.

The otherwise normal patient with an elevated cholesterol picked up in an investigation of the type recommended should, I think, be treated, although there is a risk of creating iatrogenic illness, and sufficient time has not yet elapsed to be able to say that what is hypocholesterolæmic is also antiatherogenic.

Finally, as the basis of this scheme of approach is centred around performing serum cholesterol estimations on young, healthy people, it is essential that, in order to protect them from mental trauma, they should be told that their blood is being taken for research purposes, and not because it is felt they, too, may develop coronary disease like the affected member of their family. This is not generally possible even in the larger hospitals, and the establishment of several coronary disease investigation centres working on this scheme would appear to be one of the earliest steps which should be taken by the National Heart Foundation when it begins to function.

Yours, etc.,

Royal Prince Alfred Hospital, Camperdown, N.S.W. A. M. LLOYD.
November 22, 1958.

Notes and News.

Summer Camp for Diabetic Children.

The Association of Summer Camps for Diabetic Children announces that the annual camp will be held from January 10 to 23, 1959, at Stanwell Tops, N.S.W. Any diabetic boy or girl between the ages of six and thirteen years is eligible to attend. A fully trained staff of dietitians, nursing sisters and helpers will be at the camp. Information may be obtained from the Honorary Secretary, Miss R. Pirie, c.o. Dietitian's Office, The Royal North Shore Hospital of Sydney, St. Leonards.

Gerontology and Geriatrics.

A new monthly journal has been added to the series of publications of the Excerpta Medica Foundation. This is to be known as *Gerontology and Geriatrics* and will be Section 20 of the Excerpta Medica series. The first number appeared in July, 1958. Each year the volume will contain approximately 700 pages inclusive of the yearly authors' index and a cross-referenced subject index. The object of the publication, which has been made possible in cooperation with the National Heart Institute and the National Institute of Mental Health and aided by a grant from the United States Public Health Service, is to provide a regular up-to-date comprehensive service of abstracts of the world literature in the field of gerontology and geriatrics. The subscription is £5 13s. (sterling) per year. The address of the Excerpta Medica Foundation is Kalverstraat 111, Amsterdam, the Netherlands. The agents for the Foundation in Great Britain and the Commonwealth are E. and S. Livingstone Ltd., Edinburgh.

A Binder for the Journal.

Readers are reminded that a convenient loose-leaf binder is available for filing copies of THE MEDICAL JOURNAL OF AUSTRALIA. Each binder is bound in navy blue leather cloth with the Journal's name on the spine and holds 26 copies. The individual numbers are readily inserted or withdrawn as desired. The binders are available from the Australasian Medical Publishing Company Limited, Seamer Street, Glebe, at 22s. 6d. each (*plus* postage, 1s. 6d.).

The College of Radiologists of Australasia.

DIPLOMA EXAMINATIONS.

THE College of Radiologists of Australasia will be holding examinations for the diploma of the College commencing on Monday, March 2, 1959, for Part I and Part II. Part I is held in the candidate's own State, and the Part II examination will be held in Sydney. Full details and application forms are available from the office of the College, 12th Floor, 135 Macquarie Street, Sydney. Entry forms must be received at the College office in Sydney not later than January 5, 1959.

Naval, Military and Air Force.

APPOINTMENTS.

THE following appointments, changes etc. are published in the *Commonwealth of Australia Gazette*, No. 59, of October 2, 1958.

AUSTRALIAN MILITARY FORCES.

Australian Regular Army.

Royal Australian Army Medical Corps (Medical).

The Short Service Commission granted to 3/40123 Major (Temporary Lieutenant-Colonel) A. P. Hanway is extended until 30th September, 1959.

Citizen Military Forces.

Eastern Command.

Royal Australian Army Medical Corps (Medical).—5/26429 Lieutenant-Colonel K. W. H. Harris is appointed to command 1st Field Ambulance, 21st May, 1958. 2/146556 Lieutenant-Colonel A. G. Finley relinquishes command 1st Field Ambulance, 20th May, 1958, and is transferred to the Reserve of Officers (Royal Australian Army Medical Corps (Medical)) (Eastern Command), 21st May, 1958. 2/56843 Honorary Captain A. H. Gibson is appointed from the Reserve of Officers, and to be Captain (provisionally), 1st July, 1958. 2/191720 Captain (provisionally) S. M. Bell relinquishes the provisional rank of Captain, 4th August, 1958, is transferred to the Reserve of Officers (Royal Australian Army Medical Corps (Medical)) (Eastern Command), and is granted the honorary rank of Captain, 5th August, 1958.

Southern Command.

Royal Australian Army Medical Corps (Medical).—To be Colonel, 24th December, 1956: 3/157151 Lieutenant-Colonel (Temporary Colonel) H. A. Phillips, E.D. To be Temporary Lieutenant-Colonel, 28th August, 1958: 3/101016 Major R. D. Wilson. The provisional appointments of the following officers are terminated: Captains 3/101838 P. E. Campbell, 12th February, 1958, 3/101837 E. R. Moran, 23rd March, 1958, and 3/159573 A. P. Ahern, 11th June, 1958. To be Captains (provisionally): 3/101838 Peter Ellis Campbell, 13th February, 1958, 3/101837 Edward Raymond Moran, 24th March, 1958, and 3/159573 Andrew Patrick Ahern, 12th June, 1958.

Central Command.

Royal Australian Army Medical Corps (Medical).—4/31935 Major D. F. Hannon is appointed to command 3rd Field Ambulance, and to be Temporary Lieutenant-Colonel, 23rd August, 1958. 4/31904 Lieutenant-Colonel P. S. Eyles relinquishes command 3rd Field Ambulance, 22nd August, 1958. The provisional appointment of 4/32073 Captain J. S. T. Cox is terminated, 8th August, 1958. To be Captain (provisionally), 9th August, 1958: 4/32073 John Samuel Tweedale Cox.

Western Command.

Royal Australian Army Medical Corps (Medical).—5/10736 Captain G. M. Nunn ceases to be seconded whilst in the United Kingdom, 7th July, 1958. 3/129248 Captain C. R. Naylor is transferred to the Reserve of Officers (Royal Australian Army Medical Corps (Medical)) (Western Command), 4th July, 1958. To be Temporary Major, 8th July, 1958: 5/10736 Captain G. M. Nunn.

Tasmania Command.

Royal Australian Army Medical Corps (Medical).—6/15338 Lieutenant-Colonel C. W. Clarke is appointed from the Reserve of Officers, is appointed Deputy Director of Medical Services, Headquarters Tasmania Command, and to be Colonel, 1st July, 1958. 6/15311 Colonel P. Braithwaite, E.D., relinquishes the appointment of Deputy Director of Medical Services, Headquarters Tasmania Command, 30th June, 1958, and is transferred to the Reserve of Officers (Tasmania Command), 1st July, 1958.

Reserve Citizen Military Forces.

Royal Australian Army Medical Corps (Medical).

Southern Command.—The resignations of the following officers of their commissions are accepted, 30th July, 1958: Honorary Captains B. A. Rodan and P. M. Whyte. To be Honorary Captains: Gordon Joshua Mushin, 7th July, 1958, and John Richard Fliegner, 14th July, 1958. The resignation of Honorary Captain I. A. Swain of his commission is accepted, 17th August, 1958.

The following officers are placed upon the Retired List (Eastern Command) with permission to retain their rank and wear the prescribed uniform, 30th June, 1958: Lieutenant-Colonel L. S. Loewenthal and Captain P. S. G. Anderson, E.D.

The following officer is placed upon the Retired List (Tasmania Command) with permission to retain his rank and wear the prescribed uniform, 31st October, 1958: Captain (Honorary Major) C. Bidgood, E.D.

ROYAL AUSTRALIAN AIR FORCE.

Permanent Air Force.

Medical Branch.

The following officers are appointed to a permanent commission, 7th May, 1958: Squadron Leader E. H. Stephenson (0312182), Flight Lieutenant K. N. Maunder (0217162).

Post-Graduate Work.

THE POST-GRADUATE COMMITTEE IN MEDICINE IN THE UNIVERSITY OF SYDNEY.

Week-End Course in the Art of Teaching.

THE Post-Graduate Committee in Medicine in the University of Sydney announces that a full-time week-end course in the art of teaching will be held in the Scot Skirving Lecture Theatre of the Royal Prince Alfred Hospital on Saturday and Sunday, February 14 and 15, 1959, in cooperation with Dr. H. S. Wyndham, Director-General of Education, and members of his staff.

The proceedings will be concerned with modern views on the theory and practice of teaching. These will be presented in relation to the following: the formal lecture, the tutorial, teaching by questions, principles of examining, common faults in teaching, visual aids and other ancillary methods.

Attendance at the course is strictly limited to 30 members, the fee for attendance being £1 1s. Early application, enclosing remittance, should be made to the Course Secretary, The Post-Graduate Committee in Medicine, 131

DISEASES NOTIFIED IN EACH STATE AND TERRITORY OF AUSTRALIA FOR THE WEEK ENDED NOVEMBER 15, 1958.[1]

Disease.	New South Wales.	Victoria.	Queensland.	South Australia.	Western Australia.	Tasmania.	Northern Territory.	Australian Capital Territory.	Australia.
Acute Rheumatism	..	3(3)	..	..	..	..	..	..	3
Amœbiasis	..	..	..	..	..	..	..	..	..
Ancylostomiasis	..	..	..	..	..	..	..	..	..
Anthrax	..	..	..	..	..	..	..	..	..
Bilharziasis	..	..	..	..	..	..	..	..	..
Brucellosis	..	..	..	..	..	..	..	..	..
Cholera	..	..	..	..	..	..	..	..	..
Chorea (St. Vitus)	1(1)	3(2)	..	..	..	..	..	..	4
Dengue	..	..	..	..	..	..	..	..	..
Diarrhœa (Infantile)	6	5(4)	3(2)	..	..	..	4	..	18
Diphtheria	..	..	..	..	..	..	..	..	..
Dysentery (Bacillary)	..	..	..	..	4(4)	..	7	..	11
Encephalitis	..	1	..	..	..	..	..	..	1
Filariasis	..	..	..	..	..	..	..	..	..
Homologous Serum Jaundice	..	..	..	..	..	..	..	..	..
Hydatid	..	..	..	..	..	..	..	..	..
Infective Hepatitis	87(21)	26(20)	40(1)	7(4)	2	2	3	2	169
Lead Poisoning	..	..	..	..	..	..	..	..	..
Leprosy	..	..	..	..	2	..	1	..	3
Leptospirosis	..	..	1	..	..	..	..	..	1
Malaria	..	1(1)	..	..	..	..	..	..	1
Meningococcal Infection	1	1(1)	..	..	..	..	..	..	2
Ophthalmia	..	..	..	..	..	..	..	..	..
Ornithosis	..	..	..	..	..	..	..	..	..
Paratyphoid	..	..	..	..	..	..	..	..	..
Plague	..	..	..	..	..	..	..	..	..
Poliomyelitis	..	4(3)	..	1(1)	..	..	..	..	5
Puerperal Fever	..	..	..	..	..	..	..	..	..
Rubella	..	81(73)	..	8(7)	107(100)	..	..	2	198
Salmonella Infection	..	..	..	1(1)	2(2)	..	..	..	3
Scarlet Fever	6(3)	29(24)	1	8(3)	..	2	..	..	46
Smallpox	..	..	..	..	..	..	..	..	..
Tetanus	..	..	..	..	..	..	..	..	..
Trachoma	..	..	..	..	2	..	1	..	3
Trichinosis	..	..	..	..	..	..	..	..	..
Tuberculosis	27(18)	15(11)	18(6)	2(2)	5(1)	5(1)	..	..	73
Typhoid Fever	..	..	..	..	..	..	..	..	..
Typhus (Flea-, Mite- and Tick-borne)	..	..	3	..	..	..	..	..	3
Typhus (Louse-borne)	..	..	..	..	..	..	..	..	..
Yellow Fever	..	..	..	..	..	..	..	..	..

[1] Figures in parentheses are those for the metropolitan area.

Macquarie Street, Sydney. Telephones: BU 4497-8. Telegraphic address: "Postgrad Sydney."

ROYAL PRINCE ALFRED HOSPITAL: EAR, NOSE AND THROAT DEPARTMENT.

Seminar Programme, 1958-1959.

THE staff of the Ear, Nose and Throat Department will conduct a seminar on the second Saturday of every month at 8 a.m. in the Scot Skirving Lecture Theatre, Royal Prince Alfred Hospital. The main speaker will not exceed forty minutes, and there will be a discussion at the conclusion of his remarks. All medical practitioners and clinical students are invited to attend.

At the next seminar, to be held on December 13, 1958, Dr. J. H. Lancken will speak on "Tumours of the Ear".

Notice.

THE CHILDREN'S MEDICAL RESEARCH FOUNDATION OF N.S.W.

THE following is a list of donations to the Children's Medical Research Foundation of N.S.W. received from members of the medical profession in the period November 13 to 19, 1958:

Dr. F. N. Street, Dr. and Mrs. R. M. Bowman: £20.

Dr. and Mrs. N. A. Hinds: £12 12s.

Dr. C. G. Bayliss, Dr. and Mrs. Angill: £10 10s.

Dr. R. Erby (fifth donation): £10 0s. 6d.

Dr. F. Cull, Dr. J. Lee and Dr. D. Bushell, Dr. Brian R. Morey, Dr. P. A. Deck, Dr. Marshall Andrew: £5 5s.

Dr. R. M. G. Holmes, Dr. F. G. Favaloro: £3 3s.

Previously acknowledged: £7456 16s. 9d. Total received to date: £7582 0s. 3d.

AUSTRALIAN RADIATION SOCIETY (VICTORIAN BRANCH).

THE next meeting of the Victorian Branch of the Australian Radiation Society will be held on Tuesday, December 9, 1958, at the Lecture Theatre, Cancer Institute, 483 Little Lonsdale Street, Melbourne, at 5 p.m., and not 5.30 p.m., as previously announced. The programme is a lecture entitled "Immunological Tolerance and Radiation" by Dr. J. F. Loutit, Director of the Radiobiological Research Unit, Harwell.

AUSTRALIAN RADIATION SOCIETY.

Second Australasian Conference on Radiation Biology.

THE second Australasian Conference on Radiation Biology is to be held in the Public Lecture Theatre, University of Melbourne, on December 15 to 18, 1958. Guest speakers at the conference are Dr. L. H. Gray, Director of the British Empire Cancer Campaign Radiobiological Research Unit, London, and Dr. J. F. Loutit, Director of the Radiobiological Research Unit, Atomic Energy Research Establishment, Harwell. Applications from those wishing to attend should be addressed to the convener, Dr. J. H. Martin, Cancer Institute, 483 Little Lonsdale Street, Melbourne. Telephone: MU 7831.

Nominations and Elections.

THE undermentioned has applied for election as a member of the New South Wales Branch of the British Medical Association:

Walker, Geoffrey Shepherd, M.B., B.S., 1955 (Univ. Sydney), 29 Karilla Avenue, Lane Cove.

The undermentioned has applied for election as a member of the Victorian Branch of the British Medical Association:

Pose, Freda Mary, M.D. (Univ. Basle) (registered in accordance with the provisions of the *Medical (Registration) Act*, 1957 (No. 6084), 12 Sylverly Grove, Caulfield, Victoria.

Deaths.

THE following deaths have been announced:

BOOTH.—Frederick John Booth, on November 15, 1958, at Brisbane.

CLARKE.—Raleigh Clarke, on November 22, 1958, at Melbourne.

ATKINSON.—John Lee Atkinson, on November 22, 1958, at Melbourne.

Diary for the Month.

DEC. 8.—*Victorian Branch, B.M.A.: Executive Meeting.*
DEC. 9.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
DEC. 10.—Victorian Branch, B.M.A.: Council Meeting.
DEC. 11.—New South Wales Branch, B.M.A.: Branch Meeting.
DEC. 12.—New South Wales Branch, B.M.A.: Ethics Committee.
DEC. 12.—*Queensland Branch, B.M.A.: Council Meeting.*
DEC. 12.—Tasmanian Branch, B.M.A.: Branch Council.
DEC. 16.—New South Wales Branch, B.M.A.: Hospitals Committee, Medical Politics Committee.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Medical Secretary, 135 Macquarie Street, Sydney): All contract practice appointments in New South Wales. *Anti-Tuberculosis Association of New South Wales.* The Maitland Hospital.

South Australian Branch (Honorary Secretary, 80 Brougham Place, North Adelaide): *All contract practice appointments* in South Australia.

Editorial Notices.

ALL articles submitted for publication in this Journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

References to articles and books should be carefully checked. In a reference the following information should be given: surname of author, initials of author, year, full title of article, name of journal, volume, number of first page of the article. The abbreviations used for the titles of journals are those adopted by the Quarterly Cumulative Index Medicus. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

Authors submitting illustrations are asked, if possible, to provide the originals (not photographic copies) of line drawings, graphs and diagrams, and prints from the original negatives of photomicrographs. Authors who are not accustomed to preparing drawings or photographic prints for reproduction are invited to seek the advice of the Editor.

Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary is stated.

All communications should be addressed to the Editor, THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House, Seamer Street, Glebe, New South Wales. (Telephones: MW 2651-2-3.)

Members and subscribers are requested to notify the Manager, THE MEDICAL JOURNAL OF AUSTRALIA, Seamer Street, Glebe, New South Wales, without delay, of any irregularity in the delivery of this Journal. The management cannot accept any responsibility or recognize any claim arising out of non-receipt of journals unless such notification is received within one month.

SUBSCRIPTION RATES.—Medical students and others not receiving THE MEDICAL JOURNAL OF AUSTRALIA in virtue of membership of the Branches of the British Medical Association in Australia can become subscribers to the Journal by applying to the Manager or through the usual agents and booksellers. Subscriptions can commence at the beginning of any quarter and are renewable on December 31. The rate is £5 *per annum* within Australia and the British Commonwealth of Nations, and £6 10s. *per annum* within America and foreign countries, payable in advance.